EATING DISORDERED BEHAVIOUR AND DEPRESSIVE SYMPTOMATOLOGY IN WOMEN WITH TYPE II DIABETES:

A CLINIC BASED STUDY

A Thesis
submitted in partial fulfilment
of the requirements for the degree
of

MASTERS OF ARTS
in Psychology

Marie L. Webster

University of Canterbury

1996

ACKNOWLEDGMENTS

There are several people I would like to thank who assisted in some way to the production of this thesis. Firstly, Gini McIntosh who suggested this thesis topic to me. I would also like to thank the staff at the Diabetes Centre and Diabetes Life Education for all their assistance. A special thanks to Anita Stroud, my trustworthy and reliable research colleague and friend, for all her hard work and advice, Phillippa Campbell-Tie and Dr Helen Lunt, for their expert supervision and support. Thanks to my supervisor, Dr Cindy Bulik, for her time, wealth of knowledge, statistical competence and encouragement. Thanks also to Dr Tony Ward and Dr Steve Hudson for their useful information, advice and support.

Thanks to the women who participated voluntarily in this study, the Maori Education Trust, Diabetes Research and Training Centre Trust. Finally, a special thanks to my friends and family, to whom I owe my financial survival, health and sanity, particularly my family, Anna, Liz, Catherine, Janet, Sandra, Bronwyn, Michelle and Willie.

TABLE OF CONTENTS

LIST OF TABLES

ABSTRACT

A vast quantity of literature exists examining the relationship between Insulin Dependent Diabetes Mellitus (IDDM) and eating disordered symptomatology, however little is known about such behaviour among Non-insulin Dependent Diabetes Mellitus (NIDDM) populations. Obesity has been identified as a risk factor in the development of NIDDM and the treatment of choice for obese patients with type II diabetes is dieting to promote weight loss. In addition, there appears to be an association between dieting and binge eating. Such factors considered, it seems imperative to address the issue of eating disordered behaviour in NIDDM populations.

The few studies conducted in this area indicate that eating disordered behaviour may be a problem for this population. However it is not known whether this finding translates to clinic populations. Furthermore, past studies have also found high rates of depressive symptomatology among diabetic populations.

The present study aimed to assess the prevalence and severity of binge eating and depressive symptomatology and possible associations between the two, in a clinic population of women with NIDDM. Of further interest was the occurrence of dieting behaviour and the relationship between binge eating, depressive symptomatology, insulin-manipulation, obesity, glycemic control, duration of diabetes and medical illnesses.

Although both binge eating and depressive symptomatology were significantly more prevalent and severe in the diabetic population, the rate of binge eating was not as high as that found in past research. Binge eating and depressive symptomatology were also associated. Furthermore, although dieting behaviour per se was not significantly more prevalent, the diabetic group indicated significant dissatisfaction

with their current weight and body shape. In addition, both binge eating and dieting were associated with poorer glycemic control. The prevalence of insulin manipulation among NIDDM women was comparable to many studies using IDDM populations. Reasons for these findings are discussed in light of the existing literature and possible future research areas are addressed.

GLOSSARY*

Acidosis
A condition in which there is either (i) a production in the body of two abnormal acids, or (ii) a diminution in the alkali reserve of the blood.

Autoimmune Attack
The process whereby an individual develops antibodies which attack her/his own tissues.

Beta Cell
Cells in the pancreatic islets.

Glycemia
The presence of glucose in the blood.

Glycosuria
The spillage of excess glucose into the urine, an event common in uncontrolled diabetes mellitus.

Hepatic
Involving/pertaining to the liver.

Hyperglycemia
Increase in blood sugar.

Hypoglycemia
Deficiency of blood sugar.

Insulinopenia
Relative or absolute lack of insulin.

Islet Cells
Groups of specialised cells throughout the pancreas that produce three hormones; insulin, glucagon and somatostin.

Ketoacidosis
A syndrome seen in poorly controlled diabetes mellitus with 'starvation admist plenty'; despite hyperglycemia, insulin deficiency makes the excess glucose unavailable to the cells and they must rely upon lipid metabolites (ketone bodies) resulting from incomplete lipid metabolism for energy.

Ketosis
A condition in which an excessive amount of ketones are produced by the body and these accumulate in the blood stream.

Macrovascular disease
Vascular disease of the large vessels, leading to stroke, heart attack and peripheral vascular disease.

Neuropathy
A generic term referring to a disorder of the peripheral nerves.

Nephropathy
From small blood vessel damage, clinically manifested primarily in the kidney.

Polydipsia
A general term for excessive drinking or thirst.

Polyphagia
A general term for excessive eating.

Polyuria
A general term for excessive frequency/urge of urination.

Sulfonylureas
Sulfonylurea drugs are oral hypoglycemic agents used in the management of patients with diabetes mellitus.

* see Reference section for references.

CHAPTER ONE
INTRODUCTION

1.1 General Introduction

A number of psychological and psychiatric symptoms and syndromes have been associated with diabetes mellitus, including anxiety, mood, personality and eating disorders. It is beyond the scope of the present study to review all of this literature. As such, the focus of this study is limited to (subclinical) eating disorders/behaviour and depression. A vast amount of literature exists examining the relationship between Insulin Dependent Diabetes Mellitus (IDDM; defined below) and eating disorders. The literature suggests that eating disordered behaviour may develop in IDDM populations, and subclinical eating disorders, such as binge eating, may be more prevalent among this population than in controls or the general population.

Intentional insulin manipulation, for weight control or weight loss, also appears to occur at a rate worthy of concern. Such behaviour is worrisome for many reasons, particularly as it compromises glycemic control, and therefore the physical and psychological well-being of the individuals involved.

Depression also occurs at a rate higher than that of the general population in both IDDM and Non-Insulin Dependent Diabetes Mellitus (NIDDM; defined below) samples, however the cause of this is yet unknown.

Little is known about the occurrence of eating disordered behaviour amongst NIDDM samples. The few systematic studies that have been conducted indicate that such behaviour, for example binge eating, may also be a problem for this population. However many of these studies incur a number of methodological flaws and sampling biases, limiting the validity of their findings.

The focus of the present study is eating disordered behaviour (primarily, binge eating) and depressive symptomatology and, as related to illness duration, glycemic control, and obesity, in a New Zealand clinic based population of women with NIDDM.

1.2 Literature Review

1.2.1 Background to Diabetes mellitus

Description

Diabetes mellitus is a chronic endocrine syndrome characterised by inappropriate *hyperglycemia** caused from an absolute or relative deficiency of insulin production by the pancreas, causing disturbed carbohydrate metabolism with *hypoglycemia* and *glycosuria*. Secondary changes are prone to occur in the metabolism of protein and fat, the latter leading to *ketosis* and *acidosis* (Lishman, 1987), which can consequently cause damage to many parts of the body. Some of the long-term effects of diabetes, especially if left untreated include increased chance of heart attack, stroke, blindness, kidney disorders, impotence and numbness of the feet (Moore, 1995).

The classic symptoms of diabetes include *polydipsia*, *polyuria*, sudden weight loss, headaches, drowsiness, nausea and vomiting, stomach pain, sweet breath, blurred vision and thrush. However, some individuals may experience none of these symptoms (Moore, 1995).

Diagnosis and Classification

The National Diabetes Data Group (NDDG) (1987) classification has provided a framework for both diagnosis and classification of diabetes (see Table 1). This gives three independent criteria for diagnosing diabetes mellitus, pending further classification in type.

---

* Words in italics are defined in the glossary, presented at the beginning.

Table 1

Criteria for diagnosis of Diabetes Mellitus

These are diagnostic criteria for diabetes mellitus in the non-pregnant adult. Any one of the following is sufficient to make the diagnosis. 1. A random plasma glucose level > 200 mg/dL together with classic signs and symptoms, including polydipsia, polyuria, *polyphagia*, and weight loss. 2. A fasting plasma glucose level > 140 mg/dL on two measurements, independent of symptoms. 3. A fasting plasma glucose level >140 mg/dL, but an abnormal response to two 75-gram oral glucose intolerance tests as manifested by a 2-hour plasma glucose > 200 mg/dL with an intervening value > 200 mg/dL.

The historic classification of juvenile-onset versus adult-onset diabetes eroded with the realisation that both forms of diabetes may occur in any age group (Ratner, 1992). The newer classification is an attempt to define the specific diseases according to aetiologic mechanism. The disorder once described as juvenile-onset diabetes is now more appropriately named type I diabetes, or insulin dependent diabetes mellitus (IDDM), and adult-onset type II or non-insulin dependent diabetes mellitus (NIDDM). Other types of diabetes mellitus include impaired glucose intolerance and gestational diabetes mellitus.

The distinguishing features of IDDM include patients of any age, usually thin and usually have abrupt signs and symptoms with *insulinopenia* before age 40 years. As the pancreas has stopped producing insulin, these patients often are dependent upon insulin to prevent *ketoacidosis* and to sustain life.

NIDDM patients are typically older than 40 years at diagnosis, obese and have relatively few classic symptoms. Although not dependent upon exogenous insulin for survival, they may require it for hyperglycemia that persists in spite of other therapy (NDDG, 1987). Tablets are frequently used to improve insulin production and operation. Diet and exercise habits and lose of weight may also improve metabolic control (Moore, 1995). NIDDM (type II) increases considerably with age, with a systemic increase in prevalence at each decade over the sixth (Ratner, 1992). (See Table 2 for a comparison and summary of IDDM and NIDDM).

Two major problems emerge from this classification system. First, the terms IDDM and NIDDM imply that the two types differ depending on whether insulin is used to control diabetes. This is not the case, as approximately 50% of all individuals with NIDDM are treated with insulin (Martin & Quint, 1985, cited in Ratner, 1992). Second, the descriptions of type I and type II disease in no way suggest the aetiologic mechanism underlying the disorder.

Table 2

Summary and Comparison of IDDM and NIDDM

	IDDM	**NIDDM**
Clinical features Age of Onset	Usually <30 years, but can occur at any age	Usually >35 years, but can occur at any age
Onset	Often rapid	Insidious
Weight	Non obese, thin	Often obese, may be normal
Symptoms	Polydipsia, polyuria, polyphagia	Frequently not recognised or less acute in presentation
Acute Complications	Insulin reactions and ketoacidosis	Usually none
Intermediate Complications	Failure of growth and development, problems during pregnancy	Problems during pregnancy
Long term Complications	Small blood vessel problems in eyes, kidneys, nerves; large blood vessel problems in the heart, brain and feet	Large blood vessel problems in heart, brain and feet; small blood vessel problems in eyes, feet, nerves and kidneys
Epidemiology Occurrence	10-20% of all diabetics	80-90% of all diabetics
Sex Ratio	Slight male predominance	Greater prevalence in females
Seasonal Variation	Present	Unknown
Genetics Concordance in identical twins	<50%	>90%
Environmental factors	Virus, toxins, autoimmune stimulus	Obesity, nutrition
Diabetes Control Dietary Management	Essential	Essential, may suffice
Insulin	Required for all	Required for 20-30%

Modified from Galloway et al. (1988).

Epidemiology and Demographics

Diabetes mellitus affects approximately 12 million individuals in the United States, and perhaps as many as 200 million worldwide (Ratner, 1992). In New Zealand 4-5% of people of European descent and 10-15% of individuals of Maori or Pacific Island descent have diabetes (IDDM and NIDDM combined). In addition, 7,500 new cases are diagnosed each year (Diabetes New Zealand, Inc, 1984). Of these, NIDDM patients account for approximately 90% of the United States cases. Perhaps what is most alarming however, is that approximately half of such cases may be undiagnosed (Ratner, 1992).

In addition, minority populations have significantly higher relative rates of NIDDM when compared with an age-matched white population - for example, diabetes is two times more common in blacks, 2.5 times more common in Hispanics, and five times more common in Native Americans (Winter et al. 1987; Harris et al. 1987). Although obesity certainly contributes to these increased risks, black Americans are more likely to have diabetes even when adiposity and socio-economic status are controlled for (O'Brien et al. 1989). However, Cowie, Harris, Silverman, Johnson & Rust (1993) found that differences in the prevalence of NIDDM between African Americans and whites are partially, but not entirely explained by an increased prevalence of known risk factors in African Americans.

Aetiology of IDDM

It is known that type I, or IDDM, is the result of an *autoimmune attack* on the *beta cell* (Eisenbarth, Connelly & Soeldner, 1987; cited in Ratner, 1992). In addition, it has long been known that there is a genetic propensity for the occurrence of IDDM. The risk of IDDM in the general population ranges from 1 in 400 to 1 in 1000, whilst that risk substantially increases in the offspring of individuals with diabetes to

approximately 1 in 20 to 1 in 50 (Raffel & Rotter, 1985). Recent understanding of the genetics of IDDM is leading to research into the identification of markers of susceptibility for the disorder (Ratner, 1992).

It is also clear that not all individuals at genetic risk will develop IDDM. Convincing evidence that genetics are not sufficient for the development of the disease is the finding of a 50% discordance rate of IDDM between identical twins. This suggests that some trigger is necessary for the expression of this genetic propensity. Environmental triggers for the development of IDDM have long been suspected. In particular, it is quite evident that multiple viruses appear to trigger the subsequent immunologic response in genetically predisposed individuals who develop IDDM (Ratner, 1992).

Aetiology of NIDDM

NIDDM is a very distinct disorder when compared with IDDM. NIDDM is a heterogeneous disorder, characterised by variable plasma insulin levels associated with hyperglycemia and peripheral insulin resistance. Again, heredity plays a major role in its transmission (See Table 2). The offspring of patients with type II diabetes have a 15% chance of developing NIDDM and a 30% risk of developing impaired glucose tolerance (Raffel & Rotter, 1985). In addition, there is a greater than 90% concordance rate between monozygotic twins if one has type II diabetes, suggesting the primacy of the genetic defect in this form of disease.

Currently, multiple theories exist to explain the defects observed in NIDDM and, in sum, clinical observations would suggest several phenomena at work (Ratner, 1992). Firstly, limitation in beta cell response to hyperglycemia appears to be a cornerstone of the pathophysiology of NIDDM. Regardless of the degree of peripheral

insulin resistance, if the *islet cells* have an unlimited capacity to secrete insulin, then sufficient insulin should be available to overcome any degree of resistance. However, it is apparent that the beta cell is unable to respond appropriately to a hyperglycemic challenge (Ratner, 1992). Morphological studies have revealed an approximate 50% reduction in beta cell mass in individuals with NIDDM as compared with controls, particularly when the degree of obesity is also taken into account (Klopell et al. 1985; cited in Ratner, 1992). This concept has been referred to as glucose toxicity. In essence, it has been found that beta cells chronically exposed to hyperglycemia become progressively less efficient in responding to subsequent glucose challenges. This is a reversible phenomenon in which normalisation of ambient glucose produces a dramatic improvement in insulin secretory response to a fixed glucose challenge (Leahy, 1990).

A second hallmark of NIDDM is the presence of resistance to the biologic activity of insulin noted in both the liver and peripheral tissues, where regardless of circulating insulin levels, individuals with NIDDM have a continued *hepatic* glucose production that increases circulating glucose levels (Ratner, 1992).

Risk Factors for NIDDM

In addition to the known genetic and physiological factors outlined above, there are some individuals who are more at risk for developing NIDDM than others, including those who are overweight, over 40-45 years of age, those who had diabetes during pregnancy, giving birth to a large baby (which can be independent of diabetes during pregnancy), having a family history of diabetes, having high blood pressure, being of Maori, Pacific Island or Asian descent or being on medications which can induce diabetes (eg: steroids) (Moore, 1995).

Twenty percent above ideal body weight, corresponding to a Body Mass Index (BMI) of approximately 27-28, has been defined as a health hazard (National Institutes of Health, 1985; cited in Wing, 1993).* Approximately 75% of patients with NIDDM are obese (usually associated with a BMI of greater than 30). Clinical studies suggest that obesity allows for increased expression of the genetic propensity (Ratner, 1992). Obesity is a major factor in the development and perpetuation of diabetes. Obesity is more common in women than in men and is particularly common among ethnic minority women (Cowie et al. 1993; Kuczmarski, Flegal, Campbell & Johnson, 1994; Pi-Sunyer, 1994). Cowie et al. (1993) suggest that there may be an ethnic difference in metabolic adaptation to obesity.

1.2.2 Diabetes Management

Clinical measurement

Implementation of clinical care for the individual with diabetes is now serving as the paradigm for a chronic-disease model of health care delivery. Involving the individual in his or her health care is the first step toward implementing preventative medicine and is revolutionising the concept of health care delivery (Ratner, 1992).

Self-monitoring of blood glucose (SMOBG) technology allows for a realistic management of *glycemia* on an outpatient basis (Ratner, 1992). In addition, glycated haemoglobin determinations are an additional clinical laboratory assessment that have become invaluable in both research and clinical care. The most relevant application of glycated haemoglobin (for example HbA_1*) is the assessment of long-term glycemic control. From a research perspective, it is an indispensable

---

* Body Mass Index (BMI) is used as a measure of body fat and obesity. It is formulated using weight in kg divided by height in meters squared (Garrow & Webster, 1985).

*HbA_1 is a biochemical test which provides an index of the average blood glucose level over a period of approximately 1-3 months (Rodin, Johnson, Garfinkel, Daneman & Kenshole, 1986-7). A value of below 10% is generally regarded as indicative of adequate glycemic control (Peveler & Fairburn, 1992).

parameter in relating glycemic control and long-term diabetic complications (Goldstein et al. 1987; cited in Ratner, 1992). Multiple methodologies with varying normative values exist for measuring glycated haemoglobin (Ratner, 1992).

Treatment

Despite rapid advances in medical research exploring the outer limits of molecular biology and immunology, pharmacologic intervention in individuals with diabetes has remained virtually unchanged over the last 20 years (Ratner, 1992). Interventions with diet, exercise, *sulfonylureas*, and insulin are different only in degree from those used in the 1950's. Diet appears to play a pivotal role in the treatment of this chronic disease. The main goal in treating diabetes is to restore the body's ability to produce or use carbohydrates (glucose) (Ratner, 1992).

1.2.3 Medical and Psychological Associations

Medical

With adequate treatment of diabetes, good health can be maintained. However parts of the body which can be affected in the long term are the eyes, kidneys, heart and feet. Diabetes also increases the risks of male impotence, heart attack, stroke and birth defects. Good glycemic control is a major factor in the avoidance of such complications (Diabetes New Zealand, Inc. 1984; Lloyd, Matthews, Wing & Orchard, 1992).

These problems may affect metabolic control directly, via the neuroendocrine and physiological effects of stress, or indirectly via poor self-care. Deteriorating metabolic control then may exert a negative reciprocal effect upon emotional, psychological, and interpersonal difficulties. This downward spiral can lead to life threatening crises,

such as hypoglycemic blackouts and coma, and may accelerate the complications of diabetes (Rubin & Peyrot, 1992).

The association between medical complications and quality of life is supported by Lloyd et al. (1992), who found that those individuals who experienced *macrovascular disease* or *neuropathy* reported significantly poorer quality of life compared with those who were free from all complications, and quality of life significantly deteriorated according to the presence of multiple complications.

Psychological/Psychiatric

Regardless of the growing recognition of the profound emotional and psychosocial impact of diabetes, the literature examining these areas is sparse. However a number of associated psychological complications, including mood, anxiety, eating, drug and alcohol, personality symptomatology and/or disorders and low self-esteem, have been observed to occur more frequently in individuals with diabetes (Rodin, 1983; cited in Rodin, Johnson, Garfinkel, Daneman & Kenshole, 1986-87). Weyerer, Hewer, Pfeifer-Kurda and Dilling (1989) conducted a community study and found that for both the diabetic population and individuals with other chronic medical conditions, although mainly mild, the prevalence of psychiatric disorders was significantly higher than among a healthy control group.

In addition Popkin, Callies, Lentz, Colon and Sutherland (1988) found that 38 (51%) of the patients with long-standing type I diabetes received one or more psychiatric diagnoses, including depression, anxiety and personality disorders. Interestingly, they found that none of these disorders were related to the duration of diabetes or the presence of various complications. Rubin and Peyrot (1992) believe that the prevalence of psychopathology appeared no higher among people with diabetes than among those with other chronic diseases. However

regardless of the prevalence or severity of these problems in the diabetic population at large, those individuals who do suffer from these problems are at special risk for reduced physical and emotional well-being (Rubin and Peyrot, 1992).

A literature review examining the association between diabetes and all of the above mentioned psychiatric diagnoses is beyond the scope of this research, and thus the remainder of this literature review will be limited to eating/diet related and depressive symptomatology.

Eating/diet related problems

According to Heins and Beebe (1992), food is not only a source of nutrition, but it also holds both cultural and social significance. Society also encourages individuals to be thin and to diet to achieve this ideal (Moore, 1995; Stancin, Link & Reuter, 1989). While diet is recognised clinically as the cornerstone of diabetes therapy, especially in NIDDM, individuals with diabetes say that dietary compliance is the most difficult part of their treatment regimen. There are several possible reasons for this.

One possible reason is the cultural and social factors addressed previously. In addition, diets prescribed for diabetics have the added challenge of overcoming an abnormal metabolic response to food. Secondly, the size, composition, and timing of meals must be designed to maximise insulin efficiency and promote as near normal energy metabolism as is possible. Thirdly, individuals with diabetes, especially obese women, are encouraged to focus on their food intake and are constantly being told by health professionals to look at what they eat and to keep a reasonable body weight. Finally, weight reduction improves glycemic control and thus lessens the risk of the many associated medical complications.

Consequently, as will be discussed further in this paper, recent research suggests that eating related disorders [anorexia nervosa, bulimia nervosa, and sub-clinical variants, such as binge eating (disorder)] may be somewhat more common in specific populations of individuals with diabetes than in the general population (Wing, Marcus, Epstein, Blair & Burton, 1989; Stancin et al. 1989; Rodin & Daneman, 1992; Rodin, Craven, Littlefield, Murray & Daneman, 1991; Rodin, Daneman, Johnson, Kenshole & Garfinkel, 1985).

The reasons for this association may be multiple: the alterations in body image, the sense of autonomy and identity, self-esteem, mood regulation, and family interactions may all be possible contributory factors (Heins & Beebe, 1992). In addition, as has been described, individuals with diabetes typically have poorer metabolic control. Subsequent eating disordered behaviour such as direct metabolic effects of binge eating, self-induced vomiting, laxative abuse and noncompliance with diabetes-prescribed medications, for example, intentional insulin omission, are all behaviours that have a potentially devastating effect on the often poor metabolism (Heins & Beebe, 1992; Rubin & Peyrot, 1992). Even subclinical eating disorders can interfere with glycemic control (Wing, Norwalk, Marcus, Koeske & Finegold, 1986).

Depressive symptomatology

Lloyd et al. (1992) found that patients with medical complications such as macrovascular disease also reported greater depressive symptomatology, and that high scores on instruments measuring depressive symptomatology were also related to the presence of four or more complications.

In a community study Weyerer et al. (1989) found that both those with diabetes and other chronic medical illnesses experienced a

significantly higher prevalence of depression. They noted that the high rate of depression in the diabetic population may partly be explained by an overlap of symptoms. A number of symptoms frequently experienced by individuals with depression such as weight loss, tiredness, weakness, fatigability, confusion, dizziness, impaired judgement, anxiety, trembling and emotional upset may also occur as a result of hyper-/hypoglycaemia. A limitation of this study however is that no distinction was made between IDDM and NIDDM patients, and further as it was a cross-sectional study, no causal links between psychiatric status and diabetes can be determined.

Popkin et al. (1988) found a striking 24% lifetime prevalence rate of major depression, which could not be attributed to gender, age group, duration of illness, or the presence of various diabetic complications. It was proposed that this rate may be a function of the subtle changes in the vasculature of these patient's central nervous systems. The possible behavioural and cognitive sequelae of such changes have received little attention. The subjects used in this study however were all candidates for pancreas transplant, and therefore may have been at the more severe end of the diabetes spectrum. Therefore these results should be interpreted with caution as this population may not be representative of all diabetics in general.

In further support of the association of affective illness and diabetes, Koranyi (1979) screened 2090 unselected psychiatric clinic cases, which revealed a rate of diabetes 2.3 times greater than would be expected based on population estimates. Likewise, a retrospective chart review of 203 manic-depressive patients found diabetes in 10% of the sample compared with the expected rate, derived from epidemiological surveys, of 2% (Lilliker, 1980). Both these studies however used biased samples as they were both psychiatric and clinic selected. This may therefore be more representative of the severe end of the diabetes

spectrum, rather than representative of diabetes in the general population.

Summary

Diabetes mellitus is a chronic endocrine syndrome which affects approximately 200 million people worldwide and, in New Zealand approximately 7,500 new cases are established each year. Diabetes results from either absolute or relative deficiency of insulin production by the pancreas, and can cause damage to the body. There are many long-term effects of diabetes, especially if left untreated, including increased risk of stroke, blindness and kidney disease.

There are two main types of diabetes, Type I (also called IDDM or juvenile onset) and Type II (NIDDM or maturity onset). These two types are distinguishable in many ways including factors such as age, onset, risk factors, aetiology, complications and genetic inheritance. There are also known risk factors for the development and maintenance of Type II diabetes including being overweight, over 40-45 years, having high blood pressure and being of ethnic descent (for example, Maori or Pacific Islander).

A number of medical and psychological problems and complications may occur with diabetes. Medical complications include the long term effects mentioned above and many more, which may all have a substantial effect on overall quality of life. In addition, due to the emotional impact of diabetes, mood, anxiety, eating, drug and alcohol problems and low self-esteem may often feature. Although inconclusive, it appears that eating and depressive symptomatology may be more prevalent in diabetic populations, and worthy of further investigation. Perhaps more importantly, regardless of the severity or prevalence of these problems in the diabetic population, individuals who

present with these problems are at risk for reduced physical and emotional well-being.

1.2.4 Binge eating and diabetes

What is binge eating?

The definition of what constitutes a binge, and subsequently binge eating has changed since its original inclusion in the Diagnostic and Statistical Manual of Mental Disorders - Third Edition (DSM-III) (American Psychiatric Association (APA), 1980), where the definition was 'rapid consumption of a large amount of food in a discrete period of time, usually less than two hours'. In the DSM-III-Revised (DSM-III-R) (APA, 1987), the suggested 2-hour limit was dropped.

The DSM-IV (APA, 1994) proposes a radical addition where a feeling of lack of control during the binge is also required (Beglin & Fairburn, 1992). This may be experienced either objectively and/or subjectively. An objective binge applies to the consumption of an unusually large amount of food over a short period of time, whilst a subjective binge occurs when an objectively small or normal amount of food is consumed, but the individual may have felt out of control when consuming it, or believed it to have violated some strict dietary rule (Bulik, 1994). Following an episode of binge eating, a sense of dysphoria is also experienced by many (Beglin & Fairburn, 1992), including feelings of sadness, guilt and depression. In some cases it is followed by, or interrupted by, self-induced vomiting which relieves gastric fullness and averts weight gain (Wardle, 1987). However, as highlighted by deZwaan and Mitchell (1992) the ending of an eating binge is not punctuated and thus many patients experience difficulties recalling and labelling binge eating episodes.

There may be discrepancies between the lay and the technical uses of the term 'binge eating'. Beglin and Fairburn (1992) found that a

population of young women placed greater emphasis upon loss of control and somewhat less on the quantity of food eaten. The authors also suggest that there may be both regional and sub-cultural differences in the use of the term. As a result of the differences in the use of the word and concept of a 'binge', research in this area often lacks cohesiveness and comparability. Nevertheless, the consensus regarding the primary symptoms of binge eating are the consumption of an unusually large amount of food over a short period of time (objective binge), which is often accompanied with a sense of loss of control and subsequent fullness and dysphoria (Bulik, 1994; Beglin & Fairburn, 1992).

Dieting and Binge Eating

There is considerable evidence that dieting and bingeing co-occur (Polivy & Herman, 1985; Herman & Polivy, 1990). Bingers, in short, tend to be dieters, as is shown by several researchers. For example, Loro and Orlean's (1981) population of obese bingers were all attending a weight loss clinic.

In relation to the sequential nature of these two behaviours, Polivy and Herman (1985) propose that dieting precedes bingeing, and more specifically dieting increases the likelihood of subsequent bingeing, as is supported by both the clinical and experimental literatures. Herman and Polivy (1990) state however that as binge eating is a complex concept, and difficult to understand and explain, that the literature is not yet in a strong position to posit causes or mechanisms. In addition, although there appears to be an association between restrained eating and bingeing, to assert a clear causal link would be making some unwarranted assumptions. They conclude by stating that there is much empirical and conceptual work yet to be done.

A number of propositions exist which may explain why dieting increases the likelihood of bingeing. One of these is the physiological defence, where dieting produces weight loss, which in turn may create a state of chronic hunger. Binge eating might therefore represent the body's attempt to restore weight to a more biologically appropriate level, and appears to be a psychobiological adaptation to sub-optimal weight and food deprivation (restraint theory). This biologically appropriate level may not however correspond to the cultural or personal aspirations of the dieter (Polivy & Herman, 1985). Thus, Wardle (1987) states that compulsive eating is best understood in terms of a conflict between a biologically derived drive for food and a culturally derived drive for thinness.

In addition to this argument, one concept which has been central to the development of work on dietary restraint is Nisbett's (1972) suggestion that individuals have a 'set point' for weight which is homeostatically defended. The set point may be operating above or below the levels of weight which are deemed appropriate by cultural and medical ideals. The notion of a homeostatically defended set point has come up against considerable criticism on good empirical grounds, and few people would now claim that there is a specific and stable weight for an individual (Wardle, 1987). However, the idea that dietary restraint results in biologically derived adaptations tending to facilitate weight gain is logically distinct from the notion of set point, and this now has considerable support (Wardle, 1987).

Further research suggests that role of cognitions and situational pressures are crucial to the instigation of binge eating and dieting itself. This position should not be interpreted as a rejection of the physiological factors identified above in the control of eating, rather suggests a multi-componential view. The ways in which the

physiological and non-physiological interact to affect eating are largely unclear at present (Polivy & Herman, 1985).

Polivy & Herman (1985) believe that non-physiological aspects, namely cognitive factors, may be central determinants of intake on any given occasion as the overeating or counterregulation of restrained eaters seems to be critically mediated by cognitions. For example, as long as dieter's believe themselves to be 'in control', with their diets intact, they eat frugally, but when they believe that their diets have been violated or that they are no longer capable of controlling their intake, they overeat or even binge.

A closely related construct is the 'abstinence violation effect', in which a minor violation of a strict rule results in a cognitive appraisal of failure which itself heralds a motivational collapse (Marlatt & Gordon, 1980). In the case of a restrained eater this might be seen when she decides that eating two biscuits with coffee has ruined her diet and so she might as well abandon the whole enterprise and finish the packet (Ward, Hudson & Bulik, 1993; Wardle, 1987; Wilson, 1993). Such rigid distinctions contribute to the dieter's continuing struggle with food with additional cognitive factors such as increased preoccupation with food, weight and eating, further exacerbating the problem.

Ward, Bulik and Johnson (in press) further suggest that dieting and binge eating are associated with mental suppression. They suggest that suppression of thoughts of food, weight and body shape can lead to an increase in frequency of these thoughts. Furthermore, dietary restriction is likely to be associated with successful suppression and binge eating with failed suppression.

Psychological or emotion-based dimensions, such as feeling depressed, being bored or lonely, or experiencing food cravings, are common binge eating triggers. These types of feelings, rather than physiological factors such as hunger or prolonged dieting, are more

likely to be triggers (Bruce & Agras, 1992), further supporting the primacy of an individual's thought processes and mental state, in binge eating. Polivy and Herman (1985) conclude by stating that the causal link between dieting and bingeing can be traced to the dieter's imposition of cognitive controls.

Eating disordered behaviour in IDDM populations

Both case reports and empirical studies suggest that young women with IDDM may be at high risk for developing eating disordered behaviour. Although it is agreed upon that such disordered eating occurs considerably more often in female patients, conflicting results have been reported concerning the relative prevalence of eating disordered behaviour in diabetic women versus non-diabetic women (Striegel-Moore, Nicholson & Tamborlane, 1992). Some evidence suggests that eating disorders among women with IDDM are more common than in the general population, whereas other studies conclude that IDDM patients have prevalence rates comparable with or even less than those observed among non-diabetic populations (Fairburn, Peveler, Davies, Mann & Mayou, 1991; Birk & Spencer, 1989). These conflicting results may be attributable to methodological differences, including differences in definition, measurement of eating disorders and in sampling techniques. In addition, instruments for diagnosing eating disorders do not address the issue of insulin purging.

The literature examining the area of IDDM and eating disorders is plentiful. Studies that have attempted to assess the prevalence of eating disorders among individuals with IDDM report estimates ranging from 7-35%, with an average estimate of 18% (Rodin, et al. 1986-87; Hudson, Wentworth, Hudson & Pope, 1985; Rodin, et al. 1985; Rosmark, Berne, Holmgren, Lago, Renholm, & Sohlberg, 1986; Steel, Young, Lloyd & Clarke, 1987). These estimates far exceed those

reported for the general population of young women (1.3-5%), which has led researchers to suggest that IDDM places women at risk for developing eating disorders (Shotte & Stunkard, 1987).

Wing, et al. (1986) found that diabetic adolescents differed to controls on the EAT-26 questionnaire, ordinarily indicative of more eating pathology. However diabetic subjects scored higher only on the dieting subscale of this questionnaire, probably reflecting adherence to the diabetes dietary regimen. They also found that self-reported bulimic behaviour related to poorer glycemic control. Overall, this study indicates that adolescents with diabetes report more concern about their diet than non-diabetic control subjects.

Striegel-Moore, et al. (1992) found that both IDDM and matched controls reported minimal symptoms of eating disorders. Those IDDM patients that did, related the eating disorder symptomatology to dissatisfaction with diabetes and its impact on their lives. This study had a small sample size however, and subjects were aged 8-18 years - a very wide age range to study.

A study conducted by Stancin, et al. (1989) found that 58% of their sample of IDDM women reported episodes of binge eating, and 12% met DSM-III criteria for bulimia. In an epidemiologic study conducted by Marcus, Wing, McDermott and Dodge (1990), 9% of responders indicated disordered eating and 13% reported at least 2-3 binge eating episodes per month. Rodin, et al. (1985) found both bulimia and anorexia nervosa in 6.5% of their sample; a two-fold and six-fold increase respectively.

Peveler, Fairburn, Boller and Dunger (1992) however, found that although adolescent girls with IDDM were heavier (body weight) and dieted more intensively to control their shape and weight, clinical eating disorders were no more common than among their non-diabetic counterparts. Similarly, Powers, Malone, Coovert & Schulman (1990)

did not find a higher prevalence of eating disorders among their sample of adolescent diabetic patients. However their sample included male and female IDDM patients with an average age of approximately 15.5 years. It is now widely accepted that females account for over 90% of eating disordered behaviour (Bulik, 1994; APA, 1994). Although it is important to acknowledge the occurrence of such behaviour among males, the inclusion of male subjects may not allow for an accurate result regarding prevalence of eating disordered behaviour among IDDM patients. Additionally, their results may also reflect the low age of these young adults, in that they have not completely passed through the ages at greatest risk of developing eating disorders.

Marcus, Wing, Jawad and Orchard (1992) conducted a register-based study of IDDM women, and found that diabetic subjects were comparable, with regards to eating disordered symptomatology, to those of non-diabetic subjects in standardised samples. However eating disorder symptomatology among IDDM women is significantly associated with poorer control of diabetes. This study holds value in the sense that it is a register-based sample and had a large sample size (N=188). Existing studies have tended to rely on clinic samples that may not be representative of the population of IDDM women.

When addressing issues of severity of eating disordered behaviour, LaGreca, Schwarz and Satin (1987) found that most (85%) of their population of young women with IDDM reported at least occasional bingeing and that frequency of bingeing increased with poor glycemic control.

Powers et al. (1990) highlight two important factors that may affect results and consequent conclusions in this area of study. Firstly, the criteria used for diagnosing eating disordered behaviour may differ from study to study, and secondly, methodological instruments also

differ between studies. Both these factors result in comparative difficulties.

Insulin manipulation

Insulin manipulation to promote weight loss or avoid weight gain can result in severe and potentially life threatening disturbances in metabolic control, an aspect which has received recent attention in the literature. Reasons for insulin omission may include fear of hypoglycemia, denial of illness, needle phobia, attention from others (secondary gain), and direct purging of calories as a method for managing weight (Polonsky, Anderson, Lohrer, Aponte, Jacobson & Cole, 1994).

Survey research conducted by Stancin et al. (1989) found that nearly 40% admitted to controlling their weight by insulin purging (typically by decreasing their insulin dosage), and 13.5% reported purging by other means. In terms of resulting medical complications, bulimic symptoms were positively related to reports of hospitalisations, episodes of ketoacidosis, and psychological symptoms. Reliance on self-report measures, a small sample size, and moderate response rate (65%) limit the generalisability of these results, however.

Peveler and Fairburn et al. (1992) found that 15% of their subjects had omitted or reduced their insulin dosage to influence their shape and weight and that this was not limited to those subjects with a clinical eating disorder. This study however included both boys (51) and girls (35). A similar result of 12% was found by Rodin et al. (1991). Marcus, Wing and McDermott, et al. (1990) found that 31% of their subjects, from an epidemiologic study, reported episodes of insulin manipulation to control weight and promote weight loss.

Polonsky et al. (1994) additionally found that insulin omission in an outpatient clinic-based population is also common, and that it is not

limited to younger women. Approximately 31% of the subject sample, representing women aged 13-60 years of age, reported intentional insulin omission, but only 8.8% reported frequent omission. Of the omitters, approximately half reported omitting insulin for weight management purposes. Of the other half, feelings of being emotionally overwhelmed by diabetes was paramount in promoting insulin omission. Omitters, especially those who omitted for weight management purposes, evidenced greater psychological distress, more hypoglycemic fear, poor regimen adherence, more diabetes related hospitalisations and higher rates of associated medical complications such as retinopathy and neuropathy. In addition, perhaps not surprisingly, Biggs, Basco, Patterson and Raskin (1994) found that women who withheld insulin for weight management reasons were more likely to report current or past symptoms of anorexia or bulimia nervosa and were more likely to report lying to physicians about their degree of compliance with their diabetes regimens. The results from the Biggs et al. (1994) study however must be interpreted cautiously as subject sizes were small (N=15) and there was a high refusal rate. Given these considerations, this sample may not be representative of all women who manipulate insulin.

Interestingly, LaGreca et al. (1987) found that those individuals who reported poor glycemic control coped with overeating or bingeing by omitting insulin or by avoiding the use of insulin supplements. In a New Zealand study, Hockey, Brown, and Lunt (1993) found a 17% prevalence of insulin self-manipulation, for the purposes of weight loss in an IDDM population aged 18 to 30 years. This figure is considerably lower than that reported in overseas countries such as Britain and the United States. The reason for this difference is unexplained, although subject recruitment does differ and therefore results may not be directly

comparable. Those who did self-manipulate insulin tended to have a poorer glycemic control.

Overall, these data indicate that insulin withholders for weight control reasons, exhibit more symptoms associated with the spectrum of eating disorders than non-insulin withholders (Biggs et al. 1994). In addition, they suggest that the link between omission and disordered eating may be complicated by important diabetes-specific factors. Although these data cannot indicate direction of causality, they suggest that specific attitudes, especially those associated with weight preoccupation, weight dissatisfaction (LaGreca, Schwarz, Satin, Rafkin-Mervis, Enfield & Goldberg, 1990) and with feeling emotionally overwhelmed by diabetes, may be the strongest determinants of insulin omission and problematic eating patterns, such as binge eating. However weight related omission is markedly more pathological in nature than is non-weight related omission. Rodin et al. (1991) further suggest that intentional undertreatment with insulin, which causes hyperglycemia, to reduce weight and prevent weight gain, may be regarded as an equivalent to purging, with similar purpose and consequences.

Clinically, it is important that clinicians recognise, acknowledge, and respect the importance of weight issues in their IDDM patients (Polonsky et al. 1994). Hockey et al. (1993) propose that health professionals should be aware of this method of weight reduction among their IDDM patients. Biggs et al. (1994) suggest that three measures are essential to significantly reduce the risk of serious complications as a result of insulin withholding: referral to a dietitian specialising in diabetes, close supervision of insulin administration by team members, and counselling by mental health professionals. Rosmark et al. (1986) and Striegel-Moore et al. (1992) view the increased prevalence rate of eating disordered behaviour among IDDM

patients as an important factor in the development of eating disorders. In other words, IDDM may be regarded as one risk factor in the potential development of eating disorders.

Rodin and Daneman (1992) agree, stating that certain aspects of IDDM and its management may trigger the expression of an eating disorder in susceptible individuals. Required dietary restraint and weight gain related to diabetes management are the factors most likely to be implicated. There also appears a consistent finding that eating disordered symptomatology is associated with poorer blood sugar control and poorer health outcomes (Marcus, Wing, Jawad et al. 1992).

Summary

Although the prevalence of eating disordered behaviour is controversial and depends largely on the population sampled, IDDM appears a risk factor in the development of eating disorders. Furthermore, lesser degrees of disturbed eating may be more prevalent than eating disorders per se, amongst this population. Those individuals especially at risk are those who diet, intentionally manipulate insulin and/or those who are overweight and/or concerned about their weight and shape. At a minimum, females with IDDM report concern about their diet, dissatisfaction with the illness and its subsequent impact on their lives.

Binge eating, insulin purging and poorer glycemic control also appear to occur together. Clinicians and health professionals should be aware of the relationship and consider the possibility of an eating disorder in cases where glycemic control is unexplained or uncontrolled (Marcus & Wing, 1988). At the very least, what does seem conclusive is that such behaviours compromise glycemic control, resulting in poorer physical, emotional and psychological outcomes.

Binge eating and Obesity

Although Stunkard (1959) identified binge eating as a problem in the obese more that 35 years ago, it has only recently generated research interest. Telch, Agras and Rossiter (1988) found that binge eating was significantly more prevalent among the obese, increasing sharply in those individuals with BMI scores above 34. Telch et al. (1988) consider that the dietary restriction involved in very low calorie diet prescriptions for the obese, may predispose them to binge eating upon refeeding. Furthermore, there may be additional individual, familial and societal pressures for obese individuals to maintain dieting.

Keefe, Wyshogrod, Weinberger and Agras (1984) applied the DSM-III criteria for bulimia to 44 obese patients and found that 23% of the sample met full criteria, with an additional 29% meeting all but one of the major criteria. Other researchers have reported binge eating percentages ranging from 20-46% of their overweight subjects (Marcus, Wing, & Lamparski, 1985; deZwaan, Nutzinger & Schoenbeck, 1992; Gormally, Black, Daston & Rardin, 1982; Loro & Orleans, 1981; Hawkins & Clement, 1980; Marcus & Wing, 1987); however, it is not clear from these studies which definitions of binge eating were used (Telch, et al. 1988).

Spitzer, Devlin, Walsh, Hasin, Wing, Marcus, Stunkard et al. (1992) reported that Binge Eating Disorder (BED), as proposed by the DSM-IV, was relatively common among subjects attending weight loss programs (30%), but was relatively rare in the general population with only 2% meeting full criteria for BED. Similar findings were reported by Spitzer, Yanovski, Wadden, Wing, Marcus, Stunkard, Devlin et al. (1993). Thus, it is possible that those at the more severe end of the obesity spectrum, indicated by those attending weight loss programs, pose the real concern compared with community or clinic samples.

In a recent review of the literature on BED, deZwaan, Mitchell, Raymond and Spitzer (in press) concluded that obese binge eaters represent a substantial portion of the obese in weight control programs, and that they evidence more eating- and weight-related problems as well as general psychopathology compared to their non-binge eating obese counterparts, although less than their bulimic counterparts. This is also supported by Prather and Williamson (1988) whose results suggested a continuum of psychopathology severity.

Additionally, Marcus, Smith, Santelli and Kaye (1992) found that obese binge eaters had considerable depressive symptomatology and that early onset obesity, frequent weight losses, and family histories of obesity were common. Many of these characteristics were also shared by normal weight bulimic subjects, thus suggesting behavioural and emotional similarities between the two groups. However this sample was small (n=17), and the subjects were treatment seekers, perhaps producing a sample biased towards severity. Telch et al. (1988) concluded that binge eating poses a serious problem among overweight individuals.

Research findings have produced a mixed picture and have not as yet clarified the association, if any, between obesity and emotional disturbance. When data from community studies are considered, the general consensus is that psychopathology is no more common among the obese than among normal weight controls. By contrast, studies of obese individuals drawn from clinic populations have more consistently demonstrated increased psychopathology among these patient samples. For example, Black, Goldstein, and Mason (1992) found that obese patients were more likely to have a lifetime history of mood disorders, anxiety disorders, bulimia, and tobacco dependence. Additionally, the obese subjects were more likely to evidence one or more personality disorders. Regardless of the fact that overweight individuals who seek

treatment evidence more psychopathology than those who do not, Telch and Agras (1994) concluded that binge eating per se, not obesity, accounts for the observed relationship between obesity and psychopathology.

Interestingly, Telch et al. (1988) found that none of the patients included in their sample reported self-induced vomiting or the use of cathartics or diuretics for weight control. This has been supported by a number of researchers who found that purging is less frequent in obese individuals than in normal weight binge eaters and other eating disordered populations (Hudson, Pope, Wurtman, Yurgelen-Todd, Mark & Rosenthal, 1988; Wardle & Beinart, 1981; cited in deZwaan, Nutzinger et al. 1992; Marcus & Wing, 1987; Mitchell, Pyle, Eckert, Hatsukami & Soll, 1990; Marcus, Wing, Ewing, Kern, Gooding & McDermott, 1990).

Binge Eating and NIDDM

There are a number of factors suggesting a possible association between binge eating and NIDDM. Firstly, obesity has been identified as a risk factor in the development of NIDDM. Secondly, as has been highlighted, considerable evidence suggests that binge eating may occur more readily in individuals with IDDM. Finally, the treatment of choice for obese patients with Type II diabetes is dieting to promote weight loss (Guare, Wing, Marcus, Epstein, Burton & Gooding, 1989) and, as has been discussed above there appears to be an association between dieting and binge eating. These factors considered, it seems imperative to address the issue of eating disordered behaviour among NIDDM populations. However, in contrast to the burgeoning literature on eating patterns, insulin self-manipulation and binge eating in Type I diabetic populations, there have been only a few systematic studies conducted which address this issue in Type II populations.

There appears some indication of the presence of binge eating in NIDDM. Firstly, using the Binge Eating Scale (BES), Wing et al. (1989) found that 21% of the female and 9% of the male subjects with NIDDM, met criteria for a serious binge eating problem. They concluded that binge eating is a common problem in obese Type II diabetic patients.

A number of methodological flaws limit the findings and generalisability of the Wing study. First, the population sampled included males and females, and was pre-selected from a behavioural weight control population. As was noted previously in a review of the literature on binge eating disorder, deZwaan, Mitchell, Raymond and Spitzer (in press) found that obese binge eaters represent a substantial portion of the obese in weight control programs. Thus, the Wing et al. (1989) study may have obtained a high prevalence rate of binge eating as their subject cohort were attending a weight loss program. In addition, they had a small sample (36 males and 62 females) and included no comparison group. Wing et al. (1989) suggested that further study was needed in this area and that clinical interviews should be a procedural inclusion as past studies have relied too heavily on the sole use of self-report measures.

Interestingly, in the Wing study, binge eating was unrelated to weight or glycemic control in these patients, a result which differs from previous research on IDDM populations. There may be several explanations as to why binge eating is unrelated to glycemic control in this population. First, the effect of binge eating on glycemic control may be greater in patients who are insulin deficient (Type I) than those who are insulin resistant (Type II). Second, the binge behaviour of Type I diabetes may be more severe than that of Type II diabetic patients. Finally, the marked effect of binge eating on glycemic control in Type I diabetic patients may be due not only to the episodic consumption of large amounts of food but also to the omission of insulin doses, which

frequently accompanies binge eating (LaGreca et al. 1987). This latter technique for "wasting calories" would not be available to the Type II diabetic patient (even type II diabetic patients on insulin may have some endogenous insulin secretion in response to a binge) (Wing et al. 1989).

In a second study, utilising interview and self-report techniques (BES) in a population of age, sex and weight-matched, newly diagnosed NIDDM patients, Kenardy, Mensch, Bowen and Pearson (1994) found that 14% of diabetic, compared with 4% of non-diabetic subjects reported episodes of binge eating, although there was no indication of any severe binge eating pathology. However there was no difference between the two groups in the prevalence with which they met criteria for binge eating disorder. They concluded that the relatively low rate may be due to insufficient sample size and associated power. Additionally, diabetic patients with a history of binge eating were significantly heavier, had younger age at diagnosis, and had more problems with eating in response to situational and emotional cues that did diabetic patients who did not binge.

As this population was newly diagnosed NIDDM patients, the presence of abnormal eating behaviours may have been associated with increased dietary restraint following diagnosis. The study also focused on males (n=23) and females (n=27), which may affect the results. In addition, these results are unable to determine whether binge eating pathology is associated with illness duration, as is suggested by Lustman, Griffith, Clouse and Cryer (1986).

Summary

Binge eaters may represent a distinct subgroup among the obese population. Depending upon the population sampled, they may evidence more eating- and weight-related as well as general psychopathology, particularly major depression, and closely resemble

patients with bulimia nervosa in many respects. Interestingly, as observed by many researchers, a distinct difference between patients with bulimia nervosa and obese patients who binge is the infrequency with which overweight bingers purge, in comparison with bulimic patients. In addition, according to the few systematic studies that address the issue of binge eating in NIDDM populations, there appears an increase in binge eating behaviour compared with control populations, of which severity depends largely upon the sampled population. Furthermore, those who do binge may be responding to situational and/or emotional cues. Due to the paucity of literature and methodological flaws in this area much research attention is warranted.

As in most research areas, there are many methodological problems present in this literature. With few exceptions, many of the above mentioned studies use small samples (which results in a lack of statistical power and makes it difficult to detect any real effects), differing definitions of binge eating, combinations of male and female participants, subjects selected from different populations and different outcome measures. In addition, many do not utilise control groups.

Wing et al. (1986) emphasised the need for further research with clinical interviews to obtain a better understanding of eating problems among individuals with IDDM and NIDDM. It has also been noted by many researchers (Wing et al. 1986) that eating habits are often underreported in IDDM and NIDDM patients, so as they appear adherent with their diabetes regimen. These issues prevent adequate between study comparison and are in part accountable for the controversy regarding the prevalence and severity of eating disordered behaviour among IDDM and NIDDM patients.

1.2.5 Depression and diabetes

As part of the growing concern for optimal care and management of patients with diabetes mellitus the problem of depressive disturbances in these patients has been addressed. Furthermore, depression may have a special clinical relevance in diabetes through its purported association with poor glycemic control (Lustman, et al. 1986) and decreased adherence to treatment modalities (Gavard, Lustman & Clouse, in press). Inherent in this body of work is the belief that attention to the patient's emotional status (and particularly mood) is requisite to the provision of optimal care and management of diabetes (Popkin, 1989). DeGroot and Samson (unpublished observations; cited in Jacobson, 1993) have found that the presence of a psychiatric condition, such as major depression, is associated with worsened quality of life independent of the severity of diabetes complications.

Some researcher's question whether psychiatric diagnoses can be made with adequate reliability and validity in patients with concomitant medical conditions (Lustman, Amado & Wetzel, 1983). For example, some criterion symptoms for the diagnosis of depression (eg: fatigue, weight loss) are also symptoms of unstable diabetes. However Lustman, Griffith, Gavard & Clouse (1992) found that when using structured interviews and accepted psychiatric criteria, more than 90% of the depression cases were identified. They concluded that depression in medically ill patients can be detected using these instruments.

Prevalence

Historically, much of the work in this area has lacked methodological rigour, failing to include control samples or failing to differentiate between IDDM and NIDDM. The few available studies document high rates of major depression or a significantly increased mean level on depression symptom scales in diabetics. Not

surprisingly, it appears that depression often goes unappreciated, undetected and/or untreated (Popkin, 1989). For example, in a population of both IDDM and NIDDM patients, Lustman et al. (1986) found that 33% of diabetic patients (aged 21 and older) had a lifetime history of major depression. This study hinted at slightly higher rates for NIDDM patients.

Using an IDDM population, Popkin et al. (1988) observed a lifetime prevalence of 24% for major depression which could not be attributed to gender, age group, duration of illness, or the presence of various complications. This finding suggests a prevalence rate 5-6 times that of the control group or the general population. This study however used a very small sample (n=14) and unique sample characteristics (candidates for pancreas transplantation).

In NIDDM populations, current prevalence rates for depression include 21.8% (Wing et al. 1989), 27.3% (Weyerer et al. 1989) and 26.1% reported by Montague et al. (unpublished observations, 1992; cited in Lustman et al. 1992). A firm conclusion regarding whether depression is more common in type I or type II diabetes cannot be made as many studies have focused exclusively on one type. The available evidence suggests that the prevalence of depression in IDDM patients appears similar to that seen in NIDDM patients, despite important differences in the underlying pathophysiologies of the two illnesses (Lustman et al. 1992). However in one report (deGroot and Samson (unpublished observations; cited in Jacobson, 1993), the trend was toward more psychopathology (especially depression) in NIDDM patients. This trend does not appear related solely to greater obesity in NIDDM.

Wing, Marcus, Blair, Epstein and Burton (1990) found that depression symptoms were greater in obese NIDDM subjects than in their similarly obese non-diabetic spouses. To identify the role of

obesity in depression precisely, it would be useful to compare two NIDDM groups, one obese and one nonobese. No evidence is currently available to indicate that depression prevalence differences are related to medications for diabetes or for other medical conditions (Lustman et al. 1992).

Many claim that the rates of major depression in diabetic patients may prove comparable to the rates of major depression encountered in many other chronic debilitating illnesses. However although depression symptoms are, in general, more common in the medically ill, diabetes appears to be at the high end of this continuum of depressive symptomatology severity in the medically ill (Weyerer et al. 1989).

Clinical Presentation

The mean age of onset of depression is 22.1 years in IDDM patients and 28.6 years in NIDDM patients (Lustman, Griffith & Clouse, 1988). The age of onset of depression in the general population is typically 27-35 years (Weissman, Leaf, Tischler, Blazer, Karno, Livingston & Florio, 1988) which more closely resembles the NIDDM age of onset. In patients with NIDDM, the onset of depression appears to significantly precede the onset of diabetes, and the converse is true for IDDM patients (Lustman et al. 1988).

A family history of depression is significantly more common in diabetic patients with depression (30%) compared with nondepressed diabetic patients (Lustman, Clouse, Carney & Griffith, 1987). Depression in diabetes appears to be predominantly a female illness, much as it is in the general population (Robinson, Fuller & Edmeades, 1988), although Popkin et al. (1988) and Robinson et al. (1988) observed no gender difference in the prevalence of depression in IDDM and NIDDM patients, respectively. Further studies are needed to address this issue systematically.

With regards to symptomatic presentation, Lustman, et al. (1987) states that the symptomatic expression of depression in diabetes is analogous to depression in psychiatric patients without diabetes. In addition, diabetes per se (ie: diabetes in the absence of a diagnosis of depression) is associated with a slight increase in depression symptoms, generally similar to that observed in samples of patients seeking health care. Lustman and Clouse, et al. (1988) note that depression may also influence the symptomatic expression of diabetes. In a report examining the association of diabetes symptoms to glycemic control and to psychiatric illness, depression was associated independently with increased symptoms of the metabolic disease, such as polyuria and polydipsia. Furthermore, depressed and nondepressed diabetic patients remain easily distinguishable, particularly through the cognitive and neurovegetative symptoms of depression (Lustman et al. 1987) and they may have greater dissatisfaction with physical appearance (Wing et al. 1990).

Interestingly, Haire-Joshu, Heady, Thomas, Schechtman and Fisher (in press) found a strong relationship exists between diabetes and depression in smokers with diabetes. In addition, they note that the presence of depressive symptoms may inhibit smoking cessation efforts. In support, Lustman (1994) advocates that both smoking and alcohol abuse increase the risk of diabetes complications.

Course

Little is known about the course of affective illnesses in patients with diabetes or in other physically ill patients. In a 5 year follow-up study, conducted by Lustman and Griffith, et al. (1988) 79% of the total patients ill with affective disorder during the five year period experienced episodes of major depression or dysthymia. In contrast, the likelihood of symptomatic affective disorder was only 10% over the

same follow-up period in a comparison group of diabetic subjects without depression at the index evaluation.

Occurrence of depressive episodes appeared independent of diabetes complications because both the depressed and comparison groups had similar rates of neuropathy, retinopathy, and nephropathy (Lustman and Griffith et al. 1988). However Leedom, Meeham, Procci and Zeidler (1989) question this finding, as they found that diabetic patients with complications scored significantly higher, than controls or patients without complications, on two measures of depression (Beck Depression Inventory and Zung Depression Scale), particularly on the cognitive aspects of depression.

Aetiology

Lustman et al. (1986) concluded that depression in diabetes may either cause or be caused by poor glucose regulation and that the presence of any psychiatric illness was associated with poorer long-term control of the diabetes. It has also been suggested that psychiatric illnesses, such as major depression, may themselves be intrinsic 'complications' of diabetes (Jacobson, personal communication; cited in Popkin, 1989).

Such a view is similarly held by Geringer (1990) who suggests that there may be a special relationship between depression and diabetes because of the abnormalities in the hypothalamic-pituitary adrenocortical axis, particularly cortisol response, common to both diabetes and depressive disorders. Diabetic individuals without depression also show the same abnormal response to dexamethasone suppression tests as do non-diabetic individuals who are depressed (Hudson, Hudson, Rothschild, Vignati, Schatzberg & Melby, 1984). Furthermore, functional deficiencies in norepinephrine and serotonin are believed to be associated with depression (Walsh, 1995), and these

neurotransmitters have also been implicated in animal models of diabetes (MacKenzie & Trulson, 1978).

These endocrinological abnormalities and physiological associations may be consequences of the neurological and/or metabolic derangement of both disorders. Although speculative, such physiological associations could corroborate the propensity of diabetic individuals for depression but not for other psychiatric disorders found by Weyerer et al. (1989) (Gavard et al. in press).

There may also be other diabetes-specific linkages to depression. For example, irregular blood glucose control (the rapid shifts between high and low blood glucose levels), could lead to affective lability and therefore depression in genetically at-risk individuals. However, even though many patients describe the mood shifts associated with changes in glucose levels, as yet no existing evidence suggests that repeated episodes of hypo- or hyperglycaemia have a permanent effect on mood disturbance (Jacobson, 1993).

Popkin (1989) states that clinicians seem inclined to regard depression in diabetics as predominantly psychogenic or reactive in nature. This is supported by Robinson et al. (1988) who found that the presence of depression in diabetic patients was related to type of accommodation, marital status and amount of social contact. In addition, Peyrot and Rubin (1989) found that psychosocial factors accounted for the majority of the explained variance in their data, with coping styles and self-efficacy each accounting for 19%. Of further relevance, Palinkas, Barrett-Connor and Wingard (1991) concluded that depressive symptoms in individuals with Type II diabetes may be related to awareness of diabetic condition in addition to poor health.

However, given macrovascular changes in kidney and retina, and comparable changes in small blood vessels of the central nervous system, physiological factors may have a role in the aetiology of

depression in diabetics. In addition, the prospect of a biochemical relationship between diabetes mellitus and depressive illness has been suggested, emphasising the lowering of insulin resistance and glucose utilisation in major depression, and restoration with resolution of the depression (Kronfol, Greden & Carroll, 1981). Collectively, these observations indicate that the clinician must give consideration to both psychological and physiological factors in the aetiology of depression in the diabetic.

Lustman et al. (1992) thus conclude that depression in physically ill patients results from the initial occurrence or reoccurrence of depression: 1) in response to the psychosocial hardships imposed by, or intrapsychic factors related to, the medical illness; 2) as a result of biological changes directly related to the medical illness or its treatment; or 3) developing coincidentally with the medical illness, and resulting from biological, genetic, or psychological factors not caused by the medical illness.

At present, the evidence is insufficient to explain entirely the increased prevalence of depression in diabetic patients. In fact, the cited data fail to fully exclude any hypothesis or possibility and probably best support a model that incorporates multiple mechanisms. This may explain why, in some controlled studies, depression seems more severe and prevalent in diabetic samples than in other medically ill subjects.

Binge eating and depressive symptomatology

Wing et al. (1989) found that binge eating symptomatology was strongly associated with depressive symptomatology. In addition numerous researchers (de Zwaan, Seim, Specker, Pyle, Crosby & Raymond, 1992; Marcus & Wing, 1988; Marcus, Wing, Ewing et al. 1990; Wing et al. 1989; Hudson et al. 1988 and Spitzer et al. 1993)

have found that obese binge eater's exhibit higher depression scores, and are more likely to have a history of depression, than their non-binge eating counterparts. In addition, Marcus, Wing, Guare, Blair and Jawad (1992) found that a history of major depression and depressive symptomatology are common among type II diabetic patients entering a behavioural weight-control program.

Arnow, Kenardy and Agras (1992) found that bingeing appeared to be an attempt to cope with dysphoria among their population of obese individuals. Yet there is no evidence that binge eating brings significant relief from negative mood. Some subjects reported feelings of warmth and pleasure while bingeing, but feelings immediately afterward were universally characterised by guilt. Marcus, Wing and Hopkins (1988) found that binge eating was strongly associated with self-reported dysphoria, and diet-related cognitions and behaviour. In addition, obese binge eaters, like their normal weight counterparts, appear to be preoccupied with weight, hold rigid and perfectionistic attitudes about dieting, and experience more impulsivity and less self-esteem (deZwaan, et al. in press; Williamson, Kelley, Davis, Ruggiero & Blouin, 1985).

The explanation for the association between binge eating and depression remains unclear. Depressive symptoms may be secondary to binge eating (Cooper & Fairburn, 1986), or binge eating may be secondary to depression (Pope & Hudson, 1988), or the association between binge eating and depression may be mediated by other variables. In the Wing et al. (1989) study, binge eating and depression changed together and thus made it difficult to ascertain the nature of the causal connection.

Summary

Collectively, these data indicate that depression in diabetes is a common and chronic condition. Furthermore, in their review of the literature in this area, Gavard et al. (in press) suggest that diabetic individuals are at greater risk for depression than the general adult population.

The causes of depression in diabetes remain largely unknown. Depression is probably determined by multiple factors, with the aetiological algorithm varying somewhat from one clinical case to the next (Lustman, 1994). Factors to consider include genetics, psychological factors, psychosocial hardships, noncompliance with medications, physical inactivity and obesity, precipitation by medical illness, organic abnormalities and physiological factors.

It must be remembered however, that there are clear limits in the design of a lot of these studies and that gathering unbiased samples for accurate comparison of rates of disorder is extraordinarily difficult. These biases and methodological problems may interfere with the strength the presented conclusions.

1.2.6 Rationale, aim and hypotheses of the present study

The design of the present study was identical to that of Wing et al. (1989). In the Wing study, binge eating was reported in 61% of their pre-selected population. Methodological limitations of this study are addressed above. To date, it is not known whether this sample is representative of patients attending a diabetes clinic. Information on the prevalence of binge eating in a clinic population, and the possible association with other clinical and psychological features will thus be of use in the clinical and dietary management of NIDDM.

The objectives of this present investigation were threefold:

1. To assess the prevalence and severity of binge eating and depressive symptomatology, and possible associations. It was predicted that the diabetic participants would indicate greater binge eating and depressive symptomatology than the controls.
2. To evaluate the extent of dieting and dieting behaviour (for example, insulin purging and use of laxatives and vomiting for weight control or weight loss purposes). It was further predicted that the diabetic group would indicate more dieting behaviour than the controls.
3. Establishing the relationship between age, binge eating, depressive symptomatology, self-manipulation of diabetes-specific medication, obesity, glycemic control, illness duration, medical illnesses and self-monitoring of blood glucose (SMOBG) behaviour (whether one monitors their blood glucose levels). It was expected that there would be positive correlations between binge eating, depressive symptomatology, poorer glycemic control, obesity and self-manipulation of diabetes-specific medication, in the diabetic group.

CHAPTER TWO
METHOD

2.1. Recruitment

Participants were recruited after ethical approval was granted by the Southern Regional Health Authority Ethics Committee (Canterbury) (SRHA). The diabetic participants were either discharged or current patients from a Diabetes Clinic register, who had been seen at least once previously at the Clinic. From these women, those fitting the following criteria were approached for recruitment.

They were aged 60 years (inclusive) or younger, at study commencement date (14 November 1994), and had received the diagnosis of diabetes after the age of 30 years. However, if they were diagnosed before the age of 30 years, insulin treatment did not commence within one year of diagnosis. In addition, the women were all patients seen for physician review (including complications screening) between the dates of July 1992 and October 1994. A further small assortment of additional NIDDM patients meeting the above criteria were also located from co-workers referral (for example, dietitians and doctors). Essentially, the diabetic participants were a clinic based population of women with Type II diabetes.

Prior to contacting the discharged patients, their General Practitioner's (GPs) consent was obtained in an attempt to rule out those patients who were unsuitable for the purposes of this study. In addition, the GPs of the current patients were sent a letter describing the proposed study and were given the opportunity to intervene regarding patient suitability. Upon GP consent, a letter outlining the study was sent to all eligible participants, followed by a phone call some time after to arrange possible interview times.

Each of the women that consented to participate were invited to bring a female friend/relative to the interview, who did not have diabetes and was aged between 31 and 60 years. These women composed the control group [termed 'friend controls' (Wacholder, Silverman, McLaughlin & Mandel, 1992)]. The rationale for use of friend controls was to control for demographics such as sex, age and socioeconomic status. In addition, friend controls may be more convenient and inexpensive than other alternatives (Wacholder et al. 1992). The use of 'friend controls' will be discussed further in the discussion section.

Consent forms and information sheets were sent to the participants which outlined the purpose of the study, participant requirements and gave assurance of confidentiality. However, diabetic participants were informed that with their consent their individual GPs would be contacted with glycated haemoglobin results. The information and consent forms sent to the diabetic participants differed so as to accommodate this information (see appendices).

In addition, all participants were informed that should they indicate any desire to harm themselves (as indicated by item 9 on the Beck Depression Inventory) that a Clinical Psychologist was available, and would assess this risk and that GPs may be notified* . The participants were assured that they could withdraw from the study at any time, without recrimination, should the interview and/or procedures cause any discomfort and/or pain. Written informed consent was gained by the interviewers at the arranged appointment.

---

* As both the Beck Depression Inventory (BDI) and the Binge Eating Scale (BES) are non-diagnostic in their clinical and research purpose, high scores on these measures were not utilised as indication for referral. However, clinical judgement was used by the researchers.

Participation Rate

From the aforementioned criteria, there were 205 women eligible for recruitment. Thirty-one of these women were excluded for various reasons. GPs excluded 4 due to mental illness/disability or emotional instability. Seven were excluded by the researchers due to mental illness/disability. Upon qualification of diagnoses, a further 7 were excluded as they were IDDM patients, or as their diagnoses was unclear at time of study commencement. Finally, 13 possible participants were not contactable, not living in the Christchurch area or deceased. One hundred and seventy-four possible participants remained, 55 of these refused participation when approached and the remaining 119 were recruited for study. This results in a participation rate of 68.4% of those eligible.

2.2. Participants

2.2.1 Diabetic Participants

Diabetic participants were women with maturity onset diabetes (MOD) who were recruited from a Diabetes Clinic (n=119). Patients averaged 51.3 (5.9 SD) years of age, and had an average body mass index (BMI) of 33.0 (7.6 SD). Fifty-three (44.5%) patients controlled their diabetes with insulin, 49 (41.2%) with oral medication, 11 (9.2%) with a combination of insulin and oral medication and 6 (5.0%) through diet only. An average glycated haemoglobin (HbA_1), which measures glycemic control over the 2-3 months prior, of 8.5% (1.9% SD) was recorded. Of the 119 diabetic participants 104 (87.4%), 11 (9.2%) and 4 (3.4%) were of European, Maori and other ethnicities, respectively.

2.2.2 Control Participants

Control participants averaged 47.6 (9.3 SD) years of age and had an average BMI of 27.7 (5.5 SD). Seventy-four (94.9%) and 4 (5.1%) were of European and Maori ethnicities, respectively.

2.3. Materials

Measures used were the Beck Depression Inventory (BDI), the Binge Eating Scale (BES) and a semi-structured interview schedule designed specifically for this study (see appendices).

2.3.1 Standardised Tests

Beck Depression Inventory (BDI)

The BDI was originally developed by Beck, Ward, Mendelson, Mock and Erbaugh (1961). The revised version of the BDI was used in the present study to assess current (two weeks prior to interview) mood and symptoms of depression. It is one of the most widely used instruments to measure depression in both clinically diagnosed and control populations, thus enhancing the comparability of results. It is useful in measuring the severity or intensity of depression in adults and adolescents, rather than as a sole means of diagnosis.

The revised BDI (Beck, Rush, Shaw & Emery, 1979) is a 21 item self-report instrument, in multiple choice format. It assesses the presence and severity of affective, cognitive, motivational and vegetative components of depression. Each item represents a depressive symptom or attitude, such as guilt feelings, social withdrawal or loss of appetite. Each of the 21 symptoms is composed of four statements rated from 0 to 3 in increasing levels of intensity.

The scale demonstrates high reliability and validity (Beck, Steer & Garbin, 1988), such as high internal consistency alpha of 0.86 (range

0.76-0.95). Content validity indicates that the BDI reflects 8/9 DSM-III-R criteria for major depression. Concurrent validity is moderately high (range 0.59-0.76) with other instruments that measure depressive symptomatology, such as the Hamilton Rating Scale. In addition, various studies, as stated by Beck et al. (1988), show that the BDI discriminates between psychiatric and non-psychiatric patients.

The dependent measure for this study was the raw score obtained on the BDI. Cut off scores are less than 10 (none or minimal depression); 10-18 (mild-moderate depression), 19-29 (moderate to severe depression) and 30-63 (severe depression) (Beck et al. 1988).

Binge Eating Scale (BES)

Binge eating symptomatology was assessed with the BES, which is a standardised measure of assessing binge eating symptomatology (Telch & Agras, 1994). The BES was developed by Gormally, Black, Daston and Rardin in 1982 to assess binge eating in obese populations. However it also corresponds well with independent assessments of binge eating (Marcus, Wing & Hopkins, 1988). It is a 16 item self-report instrument that is designed to assess both behavioural manifestations (eg: eating large amounts of food) and cognitions surrounding a binge episode (for example, guilt). It includes questions related to preoccupation with weight and food, inability to control urges to eat, feelings of guilt and self-hatred after eating, and frequency of strict dieting.

Gormally et al. (1982) found that the BES discriminated between patients with no, moderate, or severe binge eating problems. Higher scores indicate more bulimic behaviours. In addition, Gormally et al. (1982) stated that the BES appeared to have high internal consistency. The mean BES score reported by Gormally et al. (1982) for severe binge eaters in their normative sample was 28.9. In another study using a

cut off score on the BES of greater than or equal to 27 to indicate severe binge eating problems, Marcus, Wing, and Hopkins (1988) reported that 98% of individuals whose score in the BES indicated severe binge eating problems met DSM-III criteria for bulimia, compared with none of those whose BES score indicated few problems.

In addition, this measure has been shown to be a reliable and valid instrument for assessing eating behaviours in diabetic and non-diabetic populations (Wing et al. 1989). Furthermore, scores on the BES have been shown to correlate with HbA_1 levels in type I diabetic patients (Wing, et al. 1986).

The dependent measure for this study was the raw score obtained on the BES. As there are no established cut off scores for the BES, those suggested by Marcus, Wing, and Hopkins (1988) were utilised in the current study. They are: raw score of 17 or less (little or no problem), 18-26 (moderate) and 27 or higher (severe binge eating problems).

2.3.2 Semi-structured Questionnaire

The diabetes questionnaire was designed specifically for the purposes of this study. The diabetic participants' version included questions pertaining to demographics, diabetes management, historic and current eating patterns and weight control, knowledge about diabetes and weight issues. The control participants' version was identical, however, did not include questions regarding diabetes management.

2.4 Procedure

The procedure was explained on initial telephone contact with the participants, in the information sheet provided, and/or at the arranged

appointment time, prior to obtaining written informed consent from each participant. The majority of the testing took place at the Diabetes Clinic, however, a few home visits were made for the convenience and/or comfort of the participant.

Each of the participants were interviewed individually by one of the two available interviewers. All participants' completed the BDI, the BES, and the semi-structured questionnaire. In addition, participants' weight and height were obtained. Weight was obtained on either a balance beam (obtained while participant sits on scale) or standing scale (Seca brand), both with identical calibration, with the participants' in their usual clothes without shoes. Height was measured using either one of the two available Toledo height scales, which measure vertically from the floor and have sliding measurement markers.

Glycaemic control was measured using glycated haemoglobin (HbA_1) and blood sugar readings (current) which were obtained from the diabetic participants'. HbA_1 was measured using a capillary blood sample on the Bayer DCA 2000 machine. The laboratory norm for this machine is 5.3%. Current blood sugar levels were measured using a Glucocard machine meter. Should the diabetic participants HbA_1 have been either lower than 3.5 mmol/L (indicating hypoglycemia) or higher than 20 mmol/L (indicating hyperglcemia), it was discussed with the participant, and a nurse contacted if required.

The testing took approximately 1-1.5 hours and morning or afternoon tea was offered to all participants. Data were collected between the months of November 1994 and February 1995.

Prior to testing, the procedure was piloted on six participants' (2 control participants, 2 insulin-dependent diabetic participants' and 2 non-insulin dependent diabetic participants'). Upon feedback from the participants', few alterations were made to the semi-structured questionnaire(s) and testing began.

As a check on the reliability of the coding (see appendices), an independent post-graduate research assistant, who was not involved in data collection, assessed the accuracy of the coding. Participant confidentiality was maintained. Data were entered and analysed using the JMP package (SAS, 1994).

CHAPTER THREE
RESULTS

3.1 Demographic details: diabetics

The main diabetes treatment method was insulin, followed by tablets, insulin and tablets and diet only. The majority (75.6%) of the diabetic participants had not had any hospital admissions due to diabetes. Furthermore, the majority (96.6%) of the diabetic participants reported self-monitoring of blood glucose, with most (84.9%) using a machine meter. In addition, the average duration of diabetes was 9.7 (6.7 SD) years, with a range of 1-30 years (see table 3).

Table 3

Demographic details: diabetics (N=119)

	N	(%)
Characteristic		
Main treatment method		
Insulin	53	(44.5)
Tablets	49	(41.2)
Insulin and Tablets	11	(9.24)
Diet only	6	(5.04)
# hospital admissions		
None	90	(75.6)
One	25	(21.0)
Two	2	(1.7)
Four	2	(1.7)
SMOBG behaviour		
No	4	(3.4)
Yes	115	(96.6)
# SMOBG in last week		
None	18	(15.7)
One-Four	37	(32.2)
Five-Eight	22	(19.1)
Nine-Twelve	18	(15.7)
Fourteen-Twenty-eight	21	(18.3)
SMOBG method		
machine meter	101	(84.9)
strips against bottle	15	(12.6)
machine meter and strips against bottle	2	1.7)
no method reported	1	(0.8)
	M	**(SD)**
Illness Duration	9.7	(6.7)

3.2 Current medical problems and medication use

Comparing reported medical problem(s) and medication use, using Pearson's Chi-square analyses and Fisher's two-tailed Exact Test, diabetic participants reported significantly more medical problems (not associated with diabetes) $\chi^2(1) = 7.9$; $p<.005$ and, perhaps not surprisingly, greater medication use than the controls $\chi^2(1) = 9.2$; $p<.005$. Upon breakdown of these medical problems, the diabetic

participants reported significantly higher blood pressure, as a group, than the controls, p=.005. No differences were found for the remaining medical illnesses reported (see table 4).

Table 4

Current medical problems and medication use (not associated with diabetes).

	Diabetics		**Controls**		χ^2	**p value**
	N	**(%)**	**N**	**(%)**		
Medical problem(s)	79	(66.4)	36	(46.2)	7.9	.005
Thyroid	10	(12.7)	5	(13.9)	.3	.6
Depression	4	(5.1)	3	(8.3)	.03	.9
Other psychiatric (see appendices)	3	(3.8)	-		FET	.3
High cholesterol	8	(10.1)	1	(2.8)	FET	.09
Heart (eg: angina)	12	(15.2)	2	(5.5)	FET	.05
High BP	21	(26.6)	2	(5.5)	FET	.005
Hypertension	16	(20.3)	8	(22.2)	.45	.5
Asthma	12	(15.2)	6	(16.7)	.001	.97
Other (see appendices)	36	(45.6)	17	(47.2)	1.7	.2
Use of medication	72	(60.5)	30	(38.4)	9.2	.005

3.3 Reliability Check

As stated, a reliability check* was conducted on 33% of the coded questionnaire data, by an independent research assistant. A high level of reliability was reported for all questionnaires. The BES - total and individual scores - for controls was 100%, and diabetics 99.9%. The BDI - total and individual scores - for controls was 99.9%, and diabetics

* The formula used was total items agreed/total items x 100/1.

99.9%. Finally, the coding reliability for the diabetes questionnaire was 99.6% for controls and 99.8% for diabetics.

3.4 Hypothesis One

The objective of hypothesis one was to assess the prevalence and severity of binge eating and depressive symptomatology (using the BES and BDI, respectively), and possible associations. According to the past literature in this area, it was predicted that the diabetic participants would score higher on the BDI and BES than the controls. Using the Shapiro-Wilk test for normality, the BDI and BES scores for the control and diabetic participants were found to be non-normally distributed. Therefore, non-parametric statistical analyses, or square root transformations were used. One subject (#141) did not complete the BDI and the BES due to her emotional state at the time of interview, thus n=118.

Binge eating symptomatology

The cut-off criteria scores, suggested by Marcus and Wing (1988) were used in this study as no standardised cut-off scores exist for the BES. Using the transformed variables, an ANOVA showed that the diabetic participants scored significantly higher on the BES, than the control participants ($F(1, 194)=7.6$, $p=.006$) (see table 5).

Table 5

BES total and cut-off scores for control and diabetic participants

Characteristic	Controls (n=78) **M (SD)**	Diabetics (n=118) **M (SD)**	t	p value
BES total score	4.5 (4.2)	7.4 (6.9)	7.6	.006
Cut-off scores	**N (%)**	**N (%)**		
Little-no problem (<17)	78 (100)	108 (91.5)		
Moderate problem (18-26)	-	8 (6.8)		
Severe problem (27+)	-	2 (1.7)		

Depressive symptomatology

The cut-off scores outlined by Beck et al. (1988) were used for this study. Using the transformed variables, an ANOVA showed that the diabetic participants scored significantly higher on the BDI than the control participants (F (1, 194)=9.6, p=.002) (see table 6).

Table 6

BDI total and cut off scores for diabetic and control participants

Characteristic	Controls (n=78) **M (SD)**	Diabetics (n=118) **M (SD)**	t	p value
BDI total score	5.8 (5.1)	9.0 (8.3)	9.6	.002
Cut-off scores	**N (%)**	**N (%)**		
none-minimal (<10)	60 (76.9)	80 (67.8)		
mild-moderate (10-18)	15 (19.2)	23 (19.5)		
moderate-severe (19-29)	3 (3.9)	10 (8.5)		
severe (30-63)	-	5 (4.2)		

Relation between BES and BDI

Using the transformed variables, the BDI and BES total scores (combined diabetics and controls) were significantly correlated (r=.47, p<.00005), as were the BDI and BES total scores for the diabetic (r=.49, p<.00005) and control groups (r=.35, p<.002). When analysing the association between the BDI and BES scores, using Wilcoxin Chi-square, a significant interaction was found between the BDI severity [according to the Beck et al. (1988) cut-off scores] and BES total scores for the diabetic participants $\chi^2(3)=30.1$, p<.00001. However, no significant result was found for control participants. This result suggests that a higher BDI score is associated with a higher BES score for the diabetic, but not the control participants.

3.5 Hypothesis Two

The objective of hypothesis two was to evaluate the extent to which dieting and dieting behaviour (for example, insulin purging and use of laxatives and vomiting for weight control or weight loss purposes) occurs in a clinic population of women with type II diabetes. According to the literature, there is an association between dieting and eating disordered behaviour. It was predicted that the diabetic group would indicate more dieting and eating disordered behaviour than the control group.

Satisfaction and concerns with current body weight and shape

Comparing satisfaction with current weight and body shape and concerns with current weight and shape, using Pearson's Chi-square analyses, a significant difference was found between the diabetic and control participants χ^2 (1)= 7.4; p<.02, with diabetics reporting higher dissatisfaction than controls, and controls reporting higher satisfaction than diabetics, with their current weight and body shape (see table 7).

No significant differences were found when comparing concerns, with similar numbers of diabetic and control participants reporting concerns with current weight and body shape, 74.8% and 73.1% respectively. Furthermore, similar numbers of control and diabetic participants reported their main weight concern as desiring to decrease weight, 67.9% and 69.7% respectively (see table 7).

Several other concerns were highlighted by the control and diabetic participants with regards to current weight and body shape. Of these, the main concerns indicated were health concerns, with similar numbers of diabetic and control women reporting these, and desire for a toned body was indicated by 11.8% of the diabetic participants (see table 7).

Table 7

Satisfaction and concerns with current body weight and shape

	Controls (n=78) N (%)	**Diabetics (n=119)** N (%)	χ^2	**p value**
Characteristic				
Satisfaction with current weight			7.4	.02
Dissatisfied	23 (29.5)	58 (48.7)		
Moderately satisfied	34 (43.6)	40 (33.6)		
Very satisfied	21 (26.9)	21 (17.6)		
Concerns with current weight and body shape			.2	.6
Yes	57 (73.1)	89 (74.8)		
No	21 (26.9)	28 (23.5)		
Not indicated	-	2 (1.7)		
Weight concerns			.7	.7
Not indicated	24 (30.8)	34 (28.6)		
Increase weight	1 (1.3)	2 (1.7)		
Decrease weight	53 (67.9)	83 (69.7)		
Other concerns				
Health	6 (7.7)	6 (5.0)		
Clothing	4 (5.1)	4 (3.4)		
Bone structure	3 (3.8)	-		
Other comments	1 (1.3)	-		
Toned body	2 (2.6)	14 (11.8)		
Self-esteem and confidence	-	1 (0.8)		
More stable weight	-	1 (0.8)		

Dieting Behaviour

Comparing dieting behaviour, using Pearson's Chi-square analyses and Fisher's Exact Tests, there were no differences between the diabetic and control groups on lifetime prevalence of dieting, however there was a trend towards a higher rate of dieting amongst the

diabetics. In addition, the mean age at which participants reported beginning dieting was similar, however there was a trend for diabetics to begin earlier (see table 8).

Diabetic participants reported a significantly greater lowest BMI ($F(1, 193)=16.9$, $p=.0005$), and highest BMI ($F(1, 194)=27.0$, $p=.00005$), than the controls. In addition, the diabetics also had wider ranges on these two variables. Overall, this suggests higher weights and greater weight fluctuation for the diabetic participants in comparison to the controls (see table 8).

No significant differences were found between the two groups on the lifetime prevalence of use of diet pills, vomiting and exercising for weight and shape related purposes. However, there was a trend towards the diabetic participants having a higher use of diet pills, and the controls for having a greater lifetime prevalence of exercising (see table 8).

Table 8

Dieting Behaviour

	Controls (n=78) N (%)	**Diabetics (n=119) N (%)**	χ^2	**p value**
Characteristic				
Lifetime prevalence of dieting	45 (57.7)	83 (69.7)	3.0	.08
Age at first diet	27.5 (mean) 12.4 (SD) 11.0 - 53.0 years (range)	27.0 (mean) 11.4 (SD) 5.0 - 56.0 years (range)		
# of diets in past year				
None	61 (78.2)	93 (78.2)		
One	12 (15.4)	15 (12.6)		
Two	2 (2.6)	4 (3.4)		
Three	-	3 (2.5)		
Four	1 (1.3)	2 (1.7)		
Five	1 (1.3)	-		
Six	1 (1.3)	1 (0.8)		
Seven	-	1 (0.8)		
BMI ranges			**t**	**p value**
Lowest	21.2 (mean) 3.0 (SD) 15.0-31.2 (range)	23.9 (mean) 5.2 (SD) 14.2-40.7 (range)	16.9	.0005
Highest	29.6 (mean) 6.2 (SD) 20.0-47.6 (range)	35.5 (mean) 8.7 (SD) 19.2-56.8 (range)	**t** 27.0 -	**p value** .00005 -
Lifetime prevalence of diet pill use	8 (10.3)	15 (12.6)	.25	.6
Lifetime prevalence of vomiting	3 (3.8)	3 (2.5)	FET	.7
Lifetime prevalence of exercising	58 (74.4)	78 (65.5)	1.7	.2

Reasons for not dieting

Comparing responses to the question "are there any particular worries or reasons that stop you from dieting?", using Pearson's Chi-square analyses, no significant difference was found between control and diabetic participants, with similar numbers of both groups indicating that there were reasons for not dieting (see table 9). Furthermore, similar numbers of control and diabetic participants reported that dieting was not necessary (17.9% and 16.0%, respectively). Other reasons for not dieting included damage to health, which was reported similarly by both groups. In addition, 10.9% of the diabetic participants reported that they did not diet because of their diabetes (see table 9).

Table 9

Reasons for not dieting

	Controls (n=78) N (%)	**Diabetics (n=119)** N (%)	χ^2	**p value**
Reasons for not dieting	39 (50.0)	60 (50.4)	.004	.95
Not necessary	14 (17.9)	19 (16.0)		
Not happy when dieting	1 (1.3)	-		
Misses food	-	2 (1.7)		
May cause illness	1 (1.3)	-		
If unwell	-	-		
Irregular periods and decreased energy	3 (3.8)	-		
Pointless	4 (5.1)	3 (2.5)		
Lack of will-power	5 (6.4)	6 (5.0)		
Low blood sugars	1 (1.3)	1 (0.8)		
Damages health	7 (9.0)	4 (3.4)		
Finances	1 (1.3)	3 (2.5)		
Diabetes	-	13 (10.9)		
Self-defeating	-	2 (1.7)		
Worry if did not stay on it	-	1 (0.8)		
Family commitments	-	1 (0.8)		
Lifestyle too busy	-	4 (3.4)		
Dieting is boring	-	1 (0.8)		
Dieting increases pressure	-	3 (2.5)		

Diabetes Knowledge

In response to the question "Do you think having diabetes affects weight?" , using Pearson's Chi-square analyses, no difference was found

between groups, with a similar number in both groups suggesting that diabetes does affect a person's weight (see table 10). Trends indicated that more diabetics than controls believed that diabetes increases weight, whilst more controls than diabetics indicated that diabetes decreases weight.

When questioned regarding other ways in which diabetes may affect a person's weight many other possibilities were indicated (see table 9). Of these other possibilities, similar numbers of the control and diabetic participants (7.7% and 10.9% respectively) indicated that obesity is a risk factor for developing diabetes. Furthermore, 11.8% of the diabetic participants reported that it is harder to lose weight when one has diabetes (see table 10).

The diabetic participants were questioned as to whether they felt that diabetic medication affects their weight. Many (30.3%) indicated that diabetes medication does affect body weight. Of these, 21.8% reported that diabetes-specific medication increases body weight (see table 10).

Table 10

Diabetes knowledge

	Controls (n=78) N (%)	**Diabetics (n=119) N (%)**	χ^2	**p value**
Characteristic				
Diabetes affect body weight	62 (79.5)	92 (77.3)	1.2	.3
Increases weight	17 (21.8)	32 (26.9)		
Decreases weight	25 (32.1)	18 (15.1)		
Other possibilities				
Big-overweight people	4 (5.1)	2 (1.7)		
Obesity is a risk factor	6 (7.7)	13 (10.9)		
Become either under- or over-weight	3 (3.8)	1 (0.8)		
Eating and lifestyle changes	8 (10.3)	2 (1.7)		
Weight fluctuations	4 (5.1)	6 (5.0)		
Small people	1 (1.3)	-		
Diabetes forces one to eat	-	7 (5.9)		
Hard to lose weight	-	14 (11.8)		
Increased appetite	-	1 (0.8)		
Has maintained weight	-	2 (1.7)		
Attitude and lifestyle	-	1 (0.8)		
Speeds up metabolism	-	1 (0.8)		
Need to lose weight as part of diabetes management	-	1 (0.8)		
Does diabetes-specific medication affects body weight?	-	36 (30.3)		
Increases weight	-	26 (21.8)		
Decreases weight	-	6 (5.0)		

Diabetes-specific medication self-manipulation

The majority of the diabetic participants (62.3%) reported that they do not always take their recommended dose of diabetes-specific medication. Of those that replied, the majority (13) indicated that they decrease their dose of medication. Several other reasons were reported for altering the dose of their medication (see table 11) of which the most common were forgetting/too busy and altering their medication due to their blood sugar levels and level of exercise. Although 10 (8.4%) had thought about altering their medication for weight/shape purposes only 8 (6.7%) reported having done so (see table 11).

Table 11

Diabetes specific-medication self-manipulation

	Diabetic participants (n=118)	
	N	**(%)**
Characteristic		
Always take recommended dose		
Yes	39	(32.8)
No	74	(62.2)
Increase recommended dose	2	(1.7)
Decrease recommended dose	13	(10.9)
Reasons		
Blood sugars and exercise	19	(16.0)
Food eaten	4	(3.4)
Forgets, too busy	40	(33.6)
If away	5	(4.2)
If consuming alcohol	1	(0.8)
Financial	2	(1.7)
Doctors advise	1	(0.8)
Weight/shape purposes		
Thought about altering medication?	10	(8.4)
Done it?	8	(6.7)
In the last month?	1	(0.8)

3.6 Hypothesis Three

The objective of hypothesis three was to establish the relationship between age, binge eating, depressive symptomatology, self-manipulation of diabetes-specific medication, obesity, glycemic control, illness duration, medical illnesses and SMOBG behaviour, within the diabetic participants. It was expected that there would be positive correlations between binge eating, depressive symptomatology, poorer

glycemic control, obesity and self-manipulation of diabetes-specific medication.

Significant correlations were found between BES score and glycemic control ($r=.28$; $p<.002$), between BES score and BMI ($r=.27$; $p<.003$), between BMI and glycemic control ($r=.30$; $p<.001$), between highest BMI and BES score ($r=.31$; $p<.0008$) and between BMI range and BES score ($r=.28$; $p<.003$). Furthermore, glycemic control was significantly related to BMI range ($r=.26$; $p< .006$). These results suggest an association between body weight, weight fluctuations, glycemic control and binge eating symptomatology. It appears that higher body weight is associated with worse glycemic control. In addition, obesity and weight fluctuations are also correlated with binge eating symptomatology. A non-significant result was found between SMOBG and BES score, indicating that binge eating symptomatology is not correlated with SMOBG behaviour.

Significant correlations were also found between BDI score and glycemic control ($r=.29$; $p<.001$), between BDI and BMI ($r=.13$; $p<.15$) and between BDI and BMI range ($r=.18$; $p<.05$). As with the BES correlations above, these results suggest that depressive symptomatology is related to glycemic control, higher body weight and weight fluctuations. A non-significant result was found between SMOBG and BDI score and BDI and highest BMI, suggesting that depressive symptomatology is not correlated with SMOBG behaviour or highest body weight.

Effects of dieting

The diabetic group was then divided into dieters and non-dieters based on response to the question "have you ever changed your eating patterns or been on a diet to lose weight?". Using ANOVAs, those individuals who reported dieting, scored significantly higher on the BES

(F(1,116)=13.6, p=.0003), had higher current BMIs (F (1,116)=27.2, p=.00001), poorer glycemic control (F (1,113)=4.6, p=.03), reported higher weights (F (1, 116)=29.9, p=.00001), and greater weight fluctuations (F (1, 115)=9.8, p=.002). These results strongly suggest that dieting behaviour is related to BMI, obesity and weight fluctuations. Furthermore, dieting also appears related to binge eating symptomatology and, to a lesser degree, to depressive symptomatology, glycemic control and SMOBG behaviour. The existence of one or more medical illnesses, as indicated by a 'yes' or 'no' response, was also correlated with the ever dieting variable (r=.23, p<.01), suggesting that dieting is related to medical illnesses. Ever dieting was not significantly related to BDI and SMOBG.

Age of first diet

Age of first diet was significantly negatively correlated with highest BMI (r=-.3, p<.005) and DCA result (r=-.26, p<.01), suggesting a negative association between age at first diet and glycemic control. Furthermore, age was negatively correlated with BES score (r=-.19, p<.04). This result is suggestive of a negative association between age and binge eating symptomatology.

Presence of Medical illnesses

The diabetic group was divided into those with, and those without medical illnesses (not associated with diabetes), based on their response to their question "do you have any medical illnesses or conditions not associated with diabetes?". The existence of either one or more medical illnesses was not significantly related to illness duration, age at first diet, SMOBG behaviour, DCA result, BES score or age. However, it was significantly related to BMI range (F (1, 115)=7.5, p=.007), highest BMI (F (1, 116)= 11.7, p=.0009), BMI (F (1, 116)=9.1, p=.003), and BDI score

(F (1, 116)=4.8, p=.03). These results strongly suggest that medical complications are related to weight, weight fluctuation and obesity, and to a lesser degree, depressive symptomatology.

Illness Duration

Illness duration was not significantly correlated with ever dieted, BMI range, highest BMI, SMOBG behaviour, BMI, BES score and age at first diet, however was significantly correlated with glycated haemoglobin result (r=.2, p<.03), suggesting an association between illness duration and glycemic control.

Self-manipulation of diabetes specific medication

Self-manipulation of diabetes-specific medication was not significantly correlated with medical illness, illness duration, age, BMI range, SMOBG behaviour, DCA results, BMI, highest BMI, BDI score and BES score, yet was significantly correlated with ever dieted (r=.76, p<.01). These results suggest that diabetes-specific medication self-manipulation is associated with dieting behaviour.

CHAPTER FOUR
DISCUSSION

This section will commence with an explanation of the results found in this study, and their relevance to the past literature. Secondly, the strengths and limitations of this study will be discussed, and finally, conclusions and possible future research areas will be addressed.

4.1 Explanation of the results

Hypothesis One

Hypothesis One aimed to assess the prevalence and severity of binge eating and depressive symptomatology, and any associations between them. It was hypothesised that the diabetic participants would score higher on the BES and BDI than the control subjects. This hypothesis was supported by the present study. Furthermore, associations were also found between binge eating and depressive symptomatology.

Binge Eating Symptomatology

The diabetics indicated a higher prevalence and severity of binge eating symptomatology, than the controls, thus supporting the proposed hypothesis. All of the control participants, 78 (100%) and the majority of the diabetic participants, 108 (91.5%) reported little or no binge eating, while 8 (6.8%) of the diabetics reported moderate binge eating and 2 (1.7%) reported severe binge eating problems. It is possible that many of the diabetic participants' responses on the BES may have reflected factors such as diet restriction in response to their recommended diabetic diet. This should be considered when interpreting this finding.

This result differs substantially from Wing et al. (1989), in which they found that 21% of the females in their sample met criteria for a serious binge eating problem. There are several possible explanations for this difference. Firstly, cultural differences may exist between the New Zealand and American interpretations of the BES. These possible differences in cultural interpretation may result in overall different scores on the BES and thus, different rates of binge eating symptomatology reported.

Secondly, there may be an actual cultural difference in the prevalence of binge eating in New Zealand and the United States, more specifically, the United States may have a higher rate of binge eating symptomatology than New Zealand. Thirdly, our population had a lower BMI, on average (33.0) than the patients in the Wing study (36.7) and as binge eating appears associated with higher body weight (Telch et al. 1988), this may partly explain the lower prevalence of binge eating found in the present study.

Fourthly, the Wing et al. (1989) study used BES criteria cut-off scores suggested by Marcus, Wing and Lamparski (1985), whilst the present study used those suggested by Marcus and Wing (1988). Using those suggested by Marcus et al. (1985) a high score on the BES is indicated by a score of 25+, whereas according to Marcus and Wing (1988) a high score is indicated by a score of 27+. Therefore, according to the different criteria used, more subjects in the Wing et al. (1989) study met the criteria for a severe binge eating problem than in the present study.

Finally, this discrepancy may be due to a sample bias. In the Wing study, subjects were participating in a weight control program, while the present study surveyed a clinic based population. Many researchers, for example deZwaan, Seim et al. (in press) highlight the

increased prevalence of eating disordered behaviour in persons who attend weight control programs.

This result is similar to that of Kenardy et al. (1994), who found a higher prevalence of binge eating in the diabetic (14%) compared to the control (4%) participants. Both the present study and the Kenardy et al. (1994) study used a clinical interview as one of their assessment measures. Wing et al. (1989) suggested that past studies have relied almost exclusively upon self-report measures to determine the prevalence of binge eating. They proposed that future studies should use clinical interviews, as well as self-report outcome measures to increase the reliability and validity of the research. The use of a clinical interview in both the present and the Kenardy et al. (1994) study may thus indicate a more reliable and valid measure of binge eating symptomatology and may partly explain the comparatively lower rates of binge eating symptomatology found in these two studies when compared to the Wing study.

One difference however, between the present study and the Kenardy et al. study was that Kenardy et al. did not find any evidence of binge eating severity amongst the diabetics. One possible explanation of this finding is that Kenardy et al. used a sample of newly-diagnosed NIDDM patients, and perhaps more severe binge eating problems are associated with longer illness duration. A further explanation is that overtime, as dietary changes are made, diabetics may feel greater diet restriction, which may be associated with a higher incidence of binge eating.

Depressive Symptomatology

Although a similar number of both the control and diabetic participants scored in the non-depressed - moderately depressed range, more diabetic than control participants reported moderate-severe

depressive symptomatology. Overall, this indicates an increased prevalence and severity of depressive symptomatology amongst the diabetic participants, thus supporting the hypothesis.

Much of the literature in this area documents high rates of major depression or a significantly increased mean level on depressive symptom scales and thus, the results of the present study support this general finding. There are several possible underlying mechanisms which may explain the association between depression and diabetes.

Geringer (1990) states that abnormalities in the hypothalamic-pituitary adrenocortical axis, particularly cortisol response, is common to diabetes and depression. Furthermore, Hudson et al. (1984) indicate that diabetic individuals without depression, and depressed non-diabetic individuals show the same abnormal response to dexamethasone suppression tests. In addition, a further possible explanation is neurotransmitter functioning. In particular, norepinephrine and serotonin deficiencies have been implicated in both depression and animal models of diabetes (MacKenzie & Trulson, 1978). Overall, this research suggests that particular neuroendocrine and physiological mechanisms may be mediating factors in the relation between depression and diabetes.

In addition, there are a number of psychosocial factors that may explain the relationship between depression and diabetes. For example, Peyrot and Rubin (1989) and Palinkas et al. (1991) suggest that coping styles and self-efficacy and awareness of their diabetic condition may all mediate the relationship between depression and diabetes. Furthermore, possible relationship problems may arise as a result of the adjustment to diabetes (Robinson et al. 1988) which may result in feelings of depression. Thus, it is possible that these factors may in part explain the relationship between depression and diabetes found in the present study.

Relation between binge eating and depressive symptomatology

As was indicated by the results, the BDI and BES total scores were significantly correlated for both control and diabetic participants, suggesting an association between binge eating and depressive symptomatology. Furthermore, depressive symptomatology severity was found to be associated with binge eating symptomatology severity, for the diabetic participants only.

Many researchers have found an association between binge eating and depression (Wing et al. 1989, Hudson et al. 1988 & Spitzer et al. 1993). Furthermore, Marcus et al. (1992) found that both current and lifetime major depression was common in type II diabetic patients entering a weight control program. Therefore, the results of the present study support this literature.

Although the association between binge eating and depressive symptomatology exists, explanations for this association remain unclear. It has been suggested that depressive symptomatology occurs secondary to binge eating (Cooper & Fairburn, 1986). Alternatively, many researchers suggest that binge eating occurs secondary to depressive symptomatology (Pope & Hudson, 1988). Furthermore, recent work suggests that the association between binge eating and depressive symptomatology is mediated by other variables (Wing et al. 1989).

Due to the nature of the present study, it is not possible to ascertain the causal nature of binge eating and depressive symptomatology. It is possible, in the present study, that binge eating and depressive symptomatology may be mediated by the presence of diabetes, or at least diabetes-specific factors (for example, diabetic complications, higher body weight, taking medication that may increase weight and difficulty losing weight due to diabetes-specific medication). This is perhaps evidenced by the significant association found between

binge eating and depressive symptomatology severity, for the diabetic participants only.

This association between severity of binge eating and depressive symptomatology may be due to a possible focus, in the diabetic participants, on dietary and body weight issues. This is a necessary part of their diabetes management, but may result in them becoming preoccupied with weight and hold rigid attitudes about dieting (Williamson et al. 1985; deZwaan et al. 1994) and experiencing more diet-related cognitions and behaviour(s) (Marcus, Wing & Hopkins, 1988), than their control counterparts. In support of this, Kenardy et al. (1994) found that their population of NIDDM patients experienced more cognitive restraint in relation to their eating behaviour(s) than the control group.

Furthermore, it has been reported by Bruce & Agras (1992) that emotion-based dimensions, such as feelings of depression and loneliness may trigger binge eating episodes. Feelings of guilt and frustration may be experienced by diabetic women in relation to their dietary and eating patterns. Furthermore, greater body weight and shape dissatisfaction was reported by the diabetic women, which may result in feelings of depression. In addition, a greater prevalence of depressive symptomatology was reported in the diabetic group. As such feelings have been found to trigger binge eating episodes (Bruce & Agras, 1992), this may explain why binge eating is more prevalent in the diabetics, and may elucidate further the association between binge eating and depressive symptomatology often evident in diabetics.

Hypothesis Two

Hypothesis two aimed to evaluate the extent to which dieting and dieting behaviour (for example, insulin purging and use of laxatives and vomiting, for weight-related purposes) occurs in a clinic population of

women with type II diabetes. It was hypothesised that the diabetic population would indicate more dieting and dieting behaviour than the control population. This hypothesis was partly supported by the present study.

Satisfaction with current weight and body shape

The results of the present study indicate that more diabetics than controls are dissatisfied with their current weight and body shape, and more controls than diabetics are satisfied with their current weight and body shape. Those that indicate concerns regarding their current weight and body shape, in both the control and diabetic groups, reported a desire to decrease body weight. Furthermore, many of the diabetic participants also felt that diabetes and diabetes-specific medication affects their weight, in particular by increasing it.

Overall, these findings may be partly explained by the social pressures encouraging women to be thin and to diet to achieve this ideal (Heins & Beebe, 1992; Stancin et al. 1989). Feelings of guilt may be experienced by the diabetic woman when failing to maintain her recommended diabetic diet, and thus dissatisfaction with her weight and body shape may result. Furthermore, frustration may also be experienced by those diabetic women who feel that diabetes and diabetes-specific medication increases their weight, thus making weight loss more difficult. In addition, as obesity is a risk factor for the development of diabetes (Moore, 1995), and higher body weights were reported by the diabetic participants, the diabetic group may thus experience greater social and health pressures to maintain a healthy body weight and become dissatisfied if this does not occur.

Dieting and dieting behaviour

The diabetics indicated a higher rate of dieting than the controls, however this result was not significant, and thus only partly supporting the hypothesis that the diabetic participants would evidence greater dieting behaviour than the controls. The diabetic participants also reported both significantly greater lowest and highest BMI and greater weight fluctuations than the control group.

Overall, there does appear to be associations between the presence of a dieting history, binge eating and weight fluctuations within the diabetic group. Firstly, although not significantly, a trend towards more dieting was highlighted in the diabetic group. Secondly, more binge eating behaviour was reported, and thirdly, greater weight fluctuations were also indicated amongst the diabetic participants. There are several possible explanations and underlying mechanisms which may explain these associations.

Firstly, these results are consistent with the notion of biologically derived adaptations secondary to dietary restraint, which may facilitate weight gain. Wardle (1987) suggests that dietary restraint results in biologically sub-optimal weight. Subsequent binge eating is seen as an attempt to restore body weight to a more biologically appropriate level (Herman & Polivy, 1990).

A further possible explanation is suggested by Telch et al. (1988). They propose that weight cycling is involved with repeated dieting, which may lead to weight gain, weight fluctuation and binge eating. In addition, 'binge food' is often high in carbohydrates and fat content (Bulik, 1994), which are types of food that may lead to weight gain. The guilt often experienced after a binge episode may result in further dietary restriction which may subsequently cause weight loss. It is possible that this largely cognitive cycle may explain the association

between dieting, binge eating and weight fluctuation found in the diabetic group.

There was no reported difference between the groups in relation to diet pill use, vomiting, and exercise for weight related purposes (weight control and weight loss). This result does not support the hypothesis that the diabetic participants would report more diet related behaviour than the control group. Similar results have been found by a number of researchers (Telch et al. 1988; Hudson et al. 1988, Marcus & Wing, 1987, Mitchell et al. 1990) where purging and use of diet pills for weight control is less frequent in overweight populations than in normal weight binge eaters. The findings of the present study may thus support the literature regarding purging behaviour(s) in overweight populations. These results also suggest that extreme forms of purging behaviour are rare among NIDDM populations.

Diabetes-specific medication self-manipulation

While the majority (62.3%) of the diabetic participants reported altering (decreasing) their medication dosage, this was mainly for reasons such as forgetting, and altering dosage according to exercise and blood sugar levels. Alteration of medication dosage due to blood sugar levels and exercise is common among diabetics and is entirely appropriate behaviour.

However, eight (6.7%) of the diabetic participants reported having decreased their recommended dose of medication for weight-related purposes. Of the eight, six used insulin, one used tablets and one used tablets and insulin to manage their diabetes. In other words, of the diabetic participants that used insulin to manage their diabetes, 6 (11.3%) manipulated their insulin for weight-related reasons. In addition, self-manipulation of diabetes-specific medication was also associated with the presence of a dieting history.

Of the two studies that have addressed binge eating and eating disordered behaviour in NIDDM patients, neither have addressed the issue of diabetes-specific medication self-manipulation. However, the issue of insulin manipulation has been addressed in relation to IDDM populations (Striegel-Moore et al. 1992; Fairburn et al. 1991; Birk & Spencer, 1989; Rodin et al. 1986-1987; Hudson et al. 1985; Steel et al. 1987). The rate of insulin-manipulation found in the present study, is comparable to many of the IDDM studies, suggesting that it may be as common in some NIDDM populations as it is in some IDDM populations.

Among IDDM populations insulin manipulation is associated with poorer glycemic control and greater psychological distress and higher rates of medical complications (Biggs et al. 1994, Polonsky et al. 1994). Such associations were not found in the present study, however with a larger sample of NIDDM insulin users such results may be found.

Overall, these results suggest that although extreme forms of purging such as diet pill use, vomiting, and exercise are rare, insulin manipulation may be a used as a means of weight control or weight loss among NIDDM women, perhaps particularly among those with a dieting history. This is a particularly worrisome finding due to the known physiological and possible psychological correlates of such behaviour.

Hypothesis Three

Hypothesis three aimed to establish the relationship between variables such as age, binge eating, depressive symptomatology, self-manipulation of diabetes-specific medication, obesity, glycemic control, illness duration, medical illnesses and SMOBG behaviour within the diabetic participants. It was predicted that there would be positive correlations between binge eating, depressive symptomatology, poorer

glycemic control, obesity and self-manipulation of diabetes-specific medication. This prediction was largely supported.

Binge eating symptomatology, BMI, highest BMI, range of BMI and glycemic control were all found to be correlated, supporting the hypothesis. These results suggest that the presence of binge eating may be related to body weight, highest body weight and weight fluctuation, as is discussed previously. This result is supported by literature that claims that binge eating is more prevalent among individuals of higher body weight (Telch et al. 1988).

Furthermore, binge eating appears associated with poorer glycemic control in the present study. This is a common finding among IDDM populations, yet in the Wing et al. (1989) study binge eating was unrelated to weight or glycemic control. This discrepancy may be due to the lower BMI average of the diabetic participants in the present study. In addition, a further explanation may be the different samples used in the two studies. Wing et al. (1989) used a sample of diabetic participants recruited from a weight loss program, while the present study used a clinic based sample of NIDDM women. It is possible that as the sample in the Wing study were attending a weight loss program that they may have made some dietary changes to promote weight loss that may have also reduced their blood sugar levels. This may partly explain why the Wing et al. (1989) study did not find an association between binge eating and glycemic control.

The presence of depressive symptomatology was also found to be related to glycemic control, BMI and BMI range. This suggests that those with higher body weight and weight fluctuations may experience greater depressive symptomatology, as discussed previously. The association between poorer glycemic control and depressive symptomatology is supported by Lustman et al. (1986) who stated that depression in diabetes may either cause or be caused by poor glucose

regulation. The reason for this association is still unknown and as these results are correlational no causal direction can be posited. These results also support the literature stating that those diabetic individuals with higher body weight may experience worse glycemic control (Moore, 1995).

Effects of dieting

Those diabetics with a history of dieting had higher current and past body weights, reported greater weight fluctuations, poorer glycemic control and reported the presence of at least one current medical illness, not associated with diabetes. As is discussed previously, this result supports the higher weights and weight fluctuation evident in dieters (Telch et al. 1988). This is also supported by the negative correlation found between age of first diet and highest BMI, suggesting that earlier dieting is associated with higher body weight. Alternatively, this result may suggest that children with higher body weight may begin dieting earlier than children with lower body weight, perhaps due to greater social and health pressures.

The finding that dieting is related to poorer glycemic control among the diabetic group, and that age of the first diet is negatively correlated with glycemic control, further support the relation between bingeing and dieting, as binge eating was also found to be correlated with poorer glycemic control. It is possible that both dieting and bingeing may have more extreme effects on diabetics, due to their already poorer metabolic control and fluctuating glucose levels.

Presence of Medical illnesses

The presence of at least one medical illness among the diabetic group was related to current body weight, highest body weight and body weight range and the presence of depressive symptomatology. This is

supported by the knowledge that weight reduction improves glycemic control and health, and thus decreases the possibility of medical complications and illness (Moore, 1995).

In relation to the association between medical illnesses and depressive symptomatology, Lloyd et al. (1992) acknowledge the poorer quality of life experienced by those individuals with concurrent diabetes and other medical problems. Quality of life was shown to deteriorate for those with more than one additional medical illness. In addition, as was discussed previously, possible physiological and/or neurological abnormalities may be common to both diabetes and other medical illnesses.

Illness Duration

Finally, there appears to be an association between illness duration and glycemic control, suggesting that a longer illness duration leads to poorer glycemic control. This is supported by the finding that over the long term, due to many factors including poorer glycemic control, diabetes increases the possibility of damage to the eyes, kidneys, feet and heart (Moore, 1995).

4.2 Strengths of the present study

A number of methodological limitations exist in the diabetes literature and the literature addressing eating disordered and depressive symptomatology in diabetes (Rubin & Peyrot, 1992). Firstly, samples are often not representative of the diabetic population at large. Secondly, outcome measures are often absent and frequently poor, such as self-report measures regularly used as the sole outcome measure. Wing et al. (1989) highlight the need for the use of clinical interviews, as was discussed previously. Furthermore, the design of most studies is seriously flawed, often not including control groups.

Some studies have also failed to distinguish between type I and II diabetes and, as has been highlighted, differences between these two populations can be profound. Finally, there are often diagnostic inconsistencies between studies.

In the present study several of these methodological problems were considered. Firstly, as the sample was a clinic based population of NIDDM women, the present study provides worthwhile information for clinicians working with diabetic patients.

Secondly, a semi-structured clinical interview was used concurrently with the BES and BDI self-report measures, overcoming the sole-use of self-report assessment measures and perhaps increasing the reliability and validity of the study. A further strength of the present study is the use of the BES, which assesses not only behavioural manifestations of binge eating, but also evaluates cognitions, such as the abstinence violation effect, that are particularly relevant to binge eating.

Furthermore, a control group was used that was matched by age, sex and to some degree social class. Wacholder et al. (1992) evaluated the use of friends as controls and a number of advantages were highlighted. Friends of cases may be a more convenient and inexpensive source of controls than other alternatives. They may also be matched on several variables, such as use of the medical system in similar ways. Moreover, biases due to social class are reduced since usually the case and friend control will be of a similar socioeconomic background.

Moreover, only type II diabetic patients were recruited. In addition, the present study was not diagnostic as such, rather evaluated the presence of binge eating and depressive symptomatology. Finally, a reliability check was conducted on the coding and entering of the data, increasing the reliability of the findings.

4.3 Limitations of the present study

However, regardless of the methodological considerations outlined above, there are still a number of limitations to the present study. Firstly, with regards to the population sampled, the results of this study cannot be generalised to all diabetic patients as a clinic based sample is not representative of all diabetic groups for many reasons, including the possibility that those diabetics attending a clinic may do so as that have more severe diabetes or additional complications. Similarly, as only women were sampled these results cannot be generalised to male populations. In addition, due to the voluntary nature of recruitment, many of those diabetic patients that experience eating disordered and/or depressive symptomatology, or those that may be sensitive to these topics, may have selected themselves out. This selection bias may have been evident in the participation rate of 68.4%, in other words, 32.6% of those eligible did not participate.

Questions related to weight and appetite were omitted from the BDI in the Wing et al. (1989) study, firstly to control for BDI scores possibly being influenced by weight and appetite factors and secondly, factors such as weight, appetite, body image and satisfaction and eating behaviour were assessed using the BES. Such omission was not undertaken in the present study and therefore BDI scores in the present study may have been influenced by weight and appetite factors.

Furthermore, Kenardy et al. (1994) screened control subjects' blood glucose levels to detect any possible elevations. Kenardy et al. (1994) also screened all participants (diabetic and controls) for alcohol problems, as alcohol has been reported to disinhibit individuals' eating (Polivy & Herman, 1976). Neither of these screenings were conducted in the present study. Therefore possible alcohol usage was not known and, if used, alcohol may have disinhibited participants eating, resulting in higher binge eating symptomatology.

In relation to the assessment measures used in this study, the semi-structured questionnaire, designed specifically for the purposes of this study, although piloted on six women prior to testing, was not standardised nor validated. Furthermore, the BDI and BES, although both highly standardised and validated, are not diagnostic instruments, thus the rates of depressive and eating disordered symptomatology reported in the present study are only indications of the presence and severity of these behaviours and cannot be considered conclusive.

In addition, no normative data or established cut-off scores exist for the BES, for example the BES has no established cut-off scores to indicate a mild binge eating problem (Wing et al. 1989). Further still, the BDI gives a reliable and valid indication of depressive symptomatology over the last two weeks only, consequently indicating only current rather than lifetime prevalence of depressive symptomatology.

With regards to the use of 'friends as controls', Wacholder et al. (1992) also state that they have a number of reservations about the use of 'friends as controls'. Namely, the sampling may be biased as sociable people (case and friend) are more likely to be selected for recruitment, than are loners. In the present study, this could have been evident by the number of the diabetic participants who refused. However, those diabetic participants that did volunteer were invited to bring a friend (to be a control participant), however, were not selected on this premise. In other words, if a consenting diabetic participant did not have a friend to bring, we still recruited the diabetic participant regardless. This may have eliminated some of the bias.

A further possible shortcoming of using friends as controls is the possibility of overmatching, since friends tend to be similar with regard to lifestyle and occupational exposures of interest (Wacholder et al. 1992). In the present study we were primarily interested in dieting

behaviour, disordered eating behaviour, such as binge eating, and depressive symptomatology. As dieting and eating disordered behaviour tend to be more prevalent among obese populations (deZwaan et al. 1994), and use of friends as controls often reduces the bias of social class, it may be possible that many of the diabetic women had obese friends that may have shared similar lifestyles including dieting and eating behaviour. This may have biased the findings of the present study.

A further limitation of this study concerns correlational statistics. Although correlational statistics are useful to ascertain a possible relation between variables, a correlation does not imply causality. Thus, the causal nature of many of the associations highlighted in this study cannot be addressed. Longitudinally designed research is necessary to address these issues.

4.4 Conclusions and future research

Conclusions

The results of the present study suggest that binge eating is both more prevalent and severe among type II diabetic women than among control women, although not as prevalent as that reported in the Wing et al. (1989) study. Current depressive symptomatology was also more prevalent among the diabetic group. An association was found between binge eating and depressive symptomatology, and for the severity of binge eating and depressive symptomatology among the diabetics only.

Dieting was not significantly more prevalent among the diabetic group, however diabetics indicated greater dissatisfaction with their current weight and body shape, perhaps due to factors such as frustration and increased social and health pressures. Diabetics did not use diet pills, vomiting and exercise, for weight-related reasons, significantly more than the controls, however, insulin manipulation was

reported in numbers comparable to many of the IDDM studies. This finding suggests that although extreme forms of dieting and purging behaviour, such as diet pill use and vomiting are not common, insulin manipulation may be used among NIDDM women for weight-related reasons. Associations were found between dieting, binge eating symptomatology, depressive symptomatology, poor glycemic control, the presence of medical illness and weight fluctuations in the diabetic group.

Overall, these results suggest that dieting and bingeing behaviour may have a more detrimental effect on diabetic women due to their already poorer metabolic control and fluctuating glucose levels. There is also strong evidence that these behaviours may result in a poorer emotional and physical quality of life among NIDDM women. Thus, clinicians working in this field should be aware of these behaviours, and their effects, among women with NIDDM.

Future Research Areas

The field of type II diabetes and eating disordered behaviour is still in its infancy and as such much research is necessary. However, several primary areas are worthy of prompt future consideration. As has been highlighted, self-manipulation of diabetes-specific medication in NIDDM occurs at a rate worthy of concern and as such the practice of self-manipulation of medication in NIDDM requires much future research attention. Furthermore, as has been noted by many researchers the current assessment tools do not address the issue of self-manipulation of diabetes-specific medication. The development of a tool that measures such behaviour is therefore necessary to assessing the full extent of this behaviour.

As is discussed, some interesting work has been conducted addressing the physiological and neurological correlates and

associations between depression and diabetes. This appears promising, and further research in this area should continue.

In the present study an association was found between binge eating and depressive symptomatology, yet the causal nature of this relationship is still poorly understood. Longitudinally designed research studies may be necessary to further address the issue.

In terms of the generalisability of these research findings, the findings of the present study suggest cross-cultural differences in the rate and severity of binge eating and depressive symptomatology. However, it is not known whether these results will translate to other cultures. Cross-cultural research in this field, as in many, is strongly lacking and is certainly an area worthy of future attention. Finally, due to the female to male ratio of both eating disordered behaviour and depressive symptomatology, it is obvious why such research has taken a female subject focus, yet, further research into these areas using male samples is also needed.

REFERENCES

American Psychiatric Association. (1980). Diagnostic and Statistical Manual of Mental Disorders (Third Edition). Washington DC. American Psychiatric Association.

American Psychiatric Association. (1987). Diagnostic and Statistical Manual of Mental Disorders (Third Edition - Revised). Washington DC. American Psychiatric Association.

American Psychiatric Association. (1994). Diagnostic and Statistical Manual of Mental Disorders (Fourth Edition). Washington DC. American Psychiatric Association.

Arnow, B., Kenardy, J., & Agras, W.S. Binge eating among the obese: A descriptive study. Journal of Behavioral Medicine, 15, 155-170.

Beck, A.T., Rush, A., Shaw, B., & Emery, G. (1979). Cognitive Therapy of Depression. New York. The Guilford Press.

Beck, A.T., Steer, R., & Garbin, M. (1988). Psychometric properties of the Beck Depression Inventory: Twenty-five years of evaluation. Clinical Psychology Review, 8, 77-100.

Beck, A.T., Ward, C.H., Mendelson, M., Mock, J., & Erbaugh, J. (1961). An inventory for measuring depression. Archives of General Psychiatry, 4, 53-63.

Beglin, S.J., & Fairburn, C.G. (1992). What Is Meant by the Term "Binge"? American Journal of Psychiatry, 149, 1, 123-124.

Biggs, M.M., Basco, M.R., Patterson, G., & Raskin, P. (1994). Insulin Withholding for Weight Control in Women with Diabetes. Diabetes Care, 17, 1186-1189.

Birk, R., & Spencer, M.L. (1989). The prevalence of anorexia nervosa, bulimia, and induced glycosuria in IDDM females. Diabetes Education, 15, 336-341.

Black, D.W., Goldstein, R.B., & Mason, E.E. (1992). Prevalence of mental disorder in 88 morbidly obese bariatric clinic patients. American Journal of Psychiatry, 149, 277-234.

Bruce, B., & Agras, W.S. (1992). Binge eating in females: A population-based investigation. International Journal of Eating Disorders, 12, 365-373.

Bulik, C. (1994). Eating Disorders: Detection and Treatment. Palmerston North. The Dunmore Press Limited.

Cooper, P.J., & Fairburn, C.G. (1986). The depressive symptoms of bulimia nervosa. British Journal of Psychiatry, 148, 268-274.

Cowie, C.C., Harris, M.I., Silverman, R.E., Johnson, E.W., & Rust, K.F. (1993). Effect of Multiple Risk Factors on Differences Between Blacks and Whites in the Prevalence of NIDDM in the United States. American Journal of Epidemiology, 137, 719-732.

deZwaan, M., & Mitchell, J.E. (1992). Binge Eating in the Obese. Annals of Medicine, 24, 303-308.

deZwaan, M., Mitchell, J.E., Raymond, N.C., & Spitzer, R.L. (in press). Binge Eating Disorder: A Review of the Literature.

deZwaan, M., Nutzinger, D.O., Schonbeck, G. (1992). Binge eating in overweight females. Comprehensive Psychiatry, 33, 1-5.

deZwaan, M., Seim, H., Specker, S.M., Pyle, R.L., Crosby, R., & Raymond, N. (in press). Eating related and general psychopathology in obese individuals with binge eating disorder.

Diabetes New Zealand Inc. (1984). Patient information pamphlets.

Fairburn, C.G., Peveler, R.C., Davis, B.A., Mann, J.I., & Mayou, R.A. (1991). The prevalence of eating disorders amongst patients with insulin-dependent diabetes - a controlled study. British Medical Journal (in press).

Galloway, J.A., et al. (1988). Diabetes Mellitus (Ninth Edition). Indianapolis. Eli Lilly Co Inc.

Garrow, J.S., & Webster, J. (1985). Quetelet's index (W/H^2) as a measure of fatness. International Journal of Obesity, 9, 147-153.

Gavard, J.A., Lustman, P.J., & Clouse, R.A. (in press). Prevalence of depression in adults with diabetes: an epidemiologic evaluation. Diabetes Care.

Geringer, E.S. (1990). Affective disorders and diabetes mellitus. In Holmes, C.S. (Ed). Neuropsychological and Behavioral Aspects of Diabetes. New York. Springer-Verlag.

Gormally, J., Black, S., Daston, S., & Rardin, D. (1982). The assessment of binge eating severity among obese individuals. Addictive Behaviours, 7, 47-55.

Gould's Medical Dictionary (Fourth Edition). (1979). United States of America. McGraw-Hill.

Guare, J.C., Wing, R.R., Marcus, M.D., Epstein, L.H., Burton, L.R., & Gooding, W.E. (1989). Analysis of Changes in Eating Behaviour and Weight Loss in Type II Diabetic Patients: Which Behaviors to Change. Diabetes Care, 12, 7, 500-503.

Haire-Joshu, D., Heady, S., Thomas, L., Schechtman, K., & Fisher, E.B. (in press). Depression Symptomatology and Smoking Among Persons with Diabetes. Research in Nursing and Health.

Harris, M.I., et al. (1987). Prevalence of diabetes and impaired glucose tolerance and plasma glucose levels in the US population aged 20-74 years. Diabetes, 36, 523-534.

Hawkins, R.C., & Clement, P.F. (1980). Development and construct validation of a self-report measure of binge eating tendencies. Addictive Behaviour, 5, 219-226.

Heins, J.M., & Beebe, C.A. (1992). Nutritional Management of Diabetes Mellitus. In Haire-Joshu D (Ed). Management of Diabetes

Mellitus: Perspectives of Care Across the Life Span. United States of America. Mosby Year Book.

Herman, C.P., & Polivy, J. (1990). From dietary restraint to binge eating: Attaching causes to effects, Appetite, 2, 123-125.

Hockey, S., Brown, L.J., & Lunt, H. (1993). Prevalence of insulin self manipulation in young women with insulin dependent diabetes. New Zealand Medical Journal, 106, 474-476.

Hudson, J.I., Hudson, M.S., Rothschild, A.J., Vignati, L., Schatzberg, A.F., & Melby, J.C. (1984). Abnormal results of dexamethasone suppression tests in nondepressed patients with diabetes mellitus. Archives of General Psychiatry, 41, 1086-1089.

Hudson, J.I., Wentworth, S.M., Hudson, M.S., Pope, H.G. (1985). Prevalence of anorexia nervosa and bulimia in young diabetic women. Journal of Clinical Psychology, 46, 88-89.

Hudson, J.L., Pope, H.G., Wurtman, J., Yurgelen-Todd, D., Mark, S., & Rosenthal, N.E. (1988). Bulimia in obese individuals: Relationship to Normal-Weight Bulimia. Journal of Nervous and Mental Disease, 176, 144-152.

Jacobson, A. (1993). Depression and Diabetes (Commentary). Diabetes Care, 16, 12, 1621-1623.

Keefe, P.H., Wyshogrod, D., Weinberger, E., & Agras, W.S. (1984). Binge eating and outcome of behavioral treatment of obesity: a preliminary report. Behavior Research and Therapy, 22, 3, 319-321.

Kenardy, J., Mensch, M., Bowen, K., & Pearson, S.A. (1994). A Comparison of Eating Behaviors in Newly Diagnosed NIDDM Patients and Case-Matched Control Subjects. Diabetes Care, 17, 10, 1197-1199.

Koenigsberg, R. (Ed). (1989). Churchills' Illustrated Medical Dictionary. New York. Churchill Livingstone.

Koranyi, E.K. (1979). Morbidity and rate of undiagnosed physical illness in a psychiatric clinic population. Archives of General Psychiatry, 36, 414-419.

Kronfol, Z., Greden, J., & Carroll, B. (1981). Psychiatric Aspects of diabetes mellitus: Diabetes and Depression. British Journal of Psychiatry, 139, 172.

Kuczmarski, R.J., Flegal, K.M., Campbell, S.M., & Johnson, C.L. (1994). Increasing prevalence of overweight among US adults. Journal of the American Medical Association, 272, 205-211.

LaGreca, A.M., Schwarz, L.T., Satin, W. (1987). Eating patterns in young women with IDDM: Another look. Diabetes Care, 10, 659-660.

LaGreca, A., Schwarz, L., Satin, W., Rafkin-Mervis, L., Enfield, G., Goldberg, R. (1990). Binge eating among women with IDDM: Associations with weight dissatisfaction, adherence and metabolic control (Abstract). Diabetes (Suppl. 1), 39:164A.

Leahy, J.L. (1990). Natural history of beta-cell dysfunction in NIDDM. Diabetes Care, 13, 992-1010.

Leedom, I.J., Meeham, W.P., Procci, W.R., & Zeidler, A. (1989). Symptoms of depression in patients with type II diabetes mellitus. Psychosomatic Medicine (Abstract), 51, 265.

Lilliker, S.L. (1980). Prevalence of diabetes in a manic-depressive population. Comprehensive Psychiatry, 21, 270-275.

Lishman, W.A. (1987). Organic Psychiatry: The Psychological Consequences of Cerebral Disorder (Second Edition). London. Blackwell Scientific Publications.

Lloyd, C.E., Matthews, K.A., Wing, R.R., Orchard, T.J. (1992). Psychosocial Factors and Complications of IDDM. The Pittsburgh Epidemiology of Diabetes Complications Study. VIII. Diabetes Care, 15, 2, 166-172.

Loro, A.D., & Orleans, C.S. (1981). Binge eating in obesity: Preliminary findings and guidelines for behavioral analysis and treatment. Addictive Behaviours, 6, 155-166,

Lustman, P.J., Amado, H., & Wetzel, R.D. (1983). Depression in diabetics: A critical appraisal. Comprehensive Psychiatry, 24, 1, 65-74.

Lustman, P.J., Clouse, R.E., Carney, R.M., & Griffith, L.S. (1987). Characteristics of depression in adults with diabetes. In: Proceedings of the National Institutes of Mental Health Conference on Mental Disorders in General Health Care Settings. Seattle, WA, 1, 127-129.

Lustman, P.J., Griffith, L.S., & Clouse, R.E. (1988). Depression in adults with diabetes: results of 5-year follow-up study. Diabetes Care, 11, 605-612.

Lustman, P.J., Griffith, L.S., Clouse, R.E., & Cryer, P.E. (1986). Psychiatric illness in diabetes mellitus: Relationship to symptoms and glucose control. Journal of Nervous and Mental Disease, 174, 12, 736-742.

Lustman, P.J., Griffith, L.S., Gavard, J.A., & Clouse, R.E. (1992). Depression in Adults with Diabetes. Diabetes Care, 15, 11, 1631-1939.

Lustman, P.J. (1994). Conclusions: From Research to Practice. Diabetes Spectrum, 7, 3, 187-189.

MacKenzie, R., & Trulson, M. (1978). Effects of insulin and streptozotocin-induced diabetes on norepinephrine metabolism in rats. Journal of Neurochemistry, 30, 205-211.

MacPherson, G. (Ed). (1992). Black's Medical Dictionary. London. A & C Black.

Marcus, M.D., Smith, D., Santelli, R., & Kaye, W. (1992). Characterisation of Eating Disordered Behavior in Obese Binge

Eaters. International Journal of Eating Disorders, 12, 3, 249-255.

Marcus, M.D., & Wing, R.R. (1987). Binge eating among the obese. Annals of Behavioral Medicine, 9, 23-27.

Marcus, M.D., & Wing, R.R. (1988). Eating Disorders in patients with diabetes mellitus. Clinical Diabetes, 6, 38-43.

Marcus, M.D., Wing, R.R., Ewing, L., Kern, E., Gooding, W., & McDermott, M. (1990). Psychiatric Disorders Among Binge Eaters. International Journal of Eating Disorders, 9, 1, 69-77.

Marcus, M.D., Wing, R.R., Guare, J., Blair, E.H., & Jawad, A. (1992). Lifetime prevalence of major depression and its effect on treatment outcome in obese type II diabetic patients. Diabetes Care, 15, 253-255.

Marcus, M.D., Wing, R.R., Hopkins, J. (1988). Obese binge eaters: Affect, cognitions and response to behavioural weight control. Journal of Consulting and Clinical Psychology, 56, 433-439.

Marcus, M.D., Wing, R.R., Jawad, A., Orchard, T.J. (1992). Eating disorders symptomatology in a registry based sample of women with insulin-dependent diabetes mellitus. International Journal of Eating Disorders, 12, 425-430.

Marcus, M.D., Wing, R.R., & Lamparski, D.M. (1985). Binge eating and dietary restraint in obese patients. Addictive Behaviour, 10, 163-168.

Marcus, M.D., Wing, R.R., McDermott, M.D., & Dodge, B.A. (1990). Disordered eating is related to glycemic control in a population-based sample of women with IDDM (Abstract). Diabetes (Suppl. 1). 39:9A.

Marlatt, G.A., & Gordon, J.R. (1980). Determinants of relapse: Implications for the maintenance of behaviour change. In Davidson, P.O., & Davidson, S.M. (Eds). Behavioural Medicine: Changing Health Lifestyles. New York. Brunner/Mazel.

Mitchell, J.E., Pyle, R.L., Eckert, E.D., Hatsukami, D., Soll, E. (1990). Bulimia Nervosa in overweight individuals. Journal of Mental Disorders, 178, 324-327.

Moore, P. (1995). Diabetes. Health Services Directory, Canterbury, 48.

National Diabetes Data Group. (1987). Classification and Diagnosis of diabetes mellitus and other categories of glucose intolerance. Diabetes, 28, 1039-1057.

Nisbett, R.E. (1972). Hunger, obesity and the ventromedial hypothalamus. Psychological Reviews, 79, 433-453.

O'Brien, T.R., et al. (1989). Are racial differences in the prevalence of diabetes in adults explained by differences in obesity. Journal of the American Medical Association, 262, 1485-1488.

Palinkas, L.A., Barrett-Connor, E., & Wingard, D.L. (1991). Type 2 diabetes and depressive symptoms in older adults: a population-based study. Diabetes Medicine, 8, 532-539.

Peveler, R.C., & Fairburn, C.G. (1992). The Treatment of Bulimia Nervosa in Patients with Diabetes Mellitus. International Journal of Eating Disorders, 11, 1, 45-53.

Peveler, R.C., Fairburn, C.G., Boller, I., Dunger, D. (1992). Eating disorders in adolescents with IDDM: a controlled study. Diabetes Care, 15, 1356-1360.

Peyrot, M., & Rubin, R.R. (1989). Determinants of depression among diabetic adults (Abstract). Diabetes (Suppl. 1), 38, 9A.

Pi-Sunyer, F.X. (1994). The fattening of America. Journal of the American Medical Association, 272, 238-239.

Polivy, J., & Herman, C.P. (1976). Effects of alcohol on eating behaviour: influence of mood and perceived intoxication. Journal of Abnormal Psychology, 85, 601-606.

Polivy, J., & Herman, C.P. (1985). Dieting and Bingeing: A Causal Analysis. American Psychologist, 40, 2, 193-201.

Polonsky, W.H., Anderson, B.J., Lohrer, P.A., Aponte, J.E., Jacobson, A.M, & Cole, C.F. (1994). Insulin omission in women with IDDM. Diabetes Care, 17, 1178-1184.

Pope, H.G., & Hudson, J.I. (1988). Is bulimia nervosa a heterogeneous disorder? Lessons from the history of medicine. International Journal of Eating Disorders, 7, 155-166.

Popkin, M.K., Callies, A.L., Lentz, R.D., Colon, E.A., Sutherland, D.E. (1988). Prevalence of major depression, simple phobia, and other psychiatric disorders in patients with long-standing type I diabetes mellitus. Archives of General Psychiatry, 45, 64-68.

Popkin, M.K. (1989). Depression and Patients with Diabetes Mellitus. In: Robinson, R.G. (1989). Depression and Coexisting Disease. New York. Igaku-Shoin Medical Publishers, Inc.

Powers, P.S., Malone, J.I., Coovert, D.L., Schulman, R.G. (1990). Insulin-dependent diabetes mellitus and eating disorders: a prevalence study. Comprehensive Psychiatry, 31, 205-210.

Prather, R.C., & Williamson, D.A. (1988). Psychopathology Associated with Bulimia, Binge Eating, and Obesity. International Journal of Eating Disorders, 7, 2, 177-184.

Raffel, L.J., & Rotter, J.I. (1985). The genetics of diabetes. Clinical Diabetes, 3, 49-54.

Ratner, R.E. (1992). Overview of Diabetes Mellitus. In Haire-Joshu D (Ed). Management of Diabetes Mellitus: Perspectives of Care Across the Life Span. United States of America. Mosby Year Book.

Reber, A.S. (1985). The Penguin Dictionary of Psychology. Great Britain. Penguin Books Ltd.

Rifkin, H., & Porte, D, Jr. (Eds). (1990) Diabetes Mellitus: Theory and Practice (Fourth Edition). United States of America. Elsevier Science Publishing Co., Inc.

Robinson, W., Fuller, J.H., & Edmeades, S.P. (1987). Depression and Diabetes. Diabetic Medicine, 5, 268-271.

Rodin, G.M., Craven, J., Littlefield, C., Murray, M., & Daneman, D. (1991). Eating disorders and intentional insulin undertreatment in adolescent females with diabetes. Psychosomatics, 32, 171-176.

Rodin, G.M., Daneman, D., Johnson, L.E., Kenshole, A., & Garfinkel, P. (1985). Anorexia nervosa and bulimia in female adolescents with insulin dependent diabetes mellitus: a systematic study. Journal of Psychiatric Research, 19, 381-384.

Rodin, G.M., & Daneman, D. (1992). Eating Disorders and IDDM: A problematic association. Diabetes Care, 15, 10, 1402-.

Rodin, G.M., Johnson, L.E., Garfinkel, P.E., Daneman, D., Kenshole, A.B. (1986-7). Eating disorders in female adolescents with insulin dependent diabetes mellitus. International Journal of Psychiatry Medicine, 16, 49-57.

Rosmark, B., Berne, C., Holmgren, S., Lago, C., Renholm, G., & Sohlberg, S. (1986). Eating disorders in patients with insulin dependent diabetes mellitus. Journal of Clinical Psychiatry, 47, 547-550.

Rubin, R.R., Peyrot, M. (1992). Psychosocial Problems and Interventions in Diabetes. A Review of the Literature. Diabetes Care, 15, 11, 1640-1657.

SAS Institute (1994). JMP, 3.0.2. Statistical Package and User's Manual. Cary, North Carolina.

Segen, J.C. (1992). The Dictionary of Modern Medicine. United Kingdom. Parthenon Publishing Group Inc.

Shotte, D.E., & Stunkard, A.J. (1987). Bulimia versus bulimic behaviour on a college campus. Journal of the American Medical Association, 258, 1213-1215.

Spitzer, R.L., Devlin, M.J., Walsh, B.T., Hasin, D., Wing, R., Marcus, M.D., Stunkard, A., Wadden, T., Yanovski, S., Agras, S., Mitchell, J., & Nonas, C. (1992). Binge eating disorder: A multisite field trial of the diagnostic criteria. International Journal of Eating Disorders, 11, 191-203.

Spitzer, R.L., Yanovski, S., Wadden, T., Wing, R., Marcus, M.D., Stunkard, A., Devlin, M., Mitchell, J., Hasin, D., & Horne, R.L. (1993). Binge Eating Disorder: Its Further Validation in a Multisite Study. International Journal of Eating Disorders, 13, 2, 137-155.

Stancin, T., Link, D.L., & Reuter, J.M. (1989). Binge Eating and Purging in Young Women With IDDM. Diabetes Care, 12, 9, 601-603.

Steel, J.M., Young, R.J., Lloyd, G.G., & Clarke, B.F. (1987). Clinically apparent eating disorders in young diabetic women: associations with painful neuropathy and other complications. British Medical Journal, 294, 859-862.

Striegel-Moore, R.H., Nicholson, T.J., & Tamborlane, W.V. (1992). Prevalence of eating disorder symptoms in preadolescent and adolescent girls with IDDM. Diabetes Care, 15, 1361-1368.

Stunkard, A.J. (1959). Eating patterns and Obesity. Psychiatric Quarterly, 33, 284-292.

Telch, C.F., & Agras, W.S. (1994). Obesity, Binge Eating and Psychopathology: Are They Related? International Journal of Eating Disorders, 15, 1, 53-61.

Telch, C.F., Agras, W.S., & Rossiter, E.M. (1988). Binge Eating Increases with Increasing Adiposity. International Journal of Eating Disorders, 7, 1, 115-119.

Wacholder, S., Silverman, D.T., McLaughlin, J.K., & Mandel, J.S. (1992). Selection of Controls in Case-Control Studies. II. Types of Controls. American Journal of Epidemiology, 135 (9), 1029-1041.

Walsh, A. (1995). Neurobiology of the Affective Disorders. In: Joyce, P.R., Romans, S.E., Ellis, P.M., & Silverstone, T.S. (1995). Affective Disorders (Chapter 11). New Zealand.

Ward, T., Hudson, S.M., & Bulik, C.M. (1993). The Abstinence Violation Effect in Bulimia Nervosa. Addictive Behaviours, 18, 671-680.

Ward, T., Bulik, C.M., & Johnson, L. (in press). Return of the Suppressed: Mental Control and Bulimia Nervosa.

Wardle, J. (1987). Compulsive Eating and dietary restraint. British Journal of Clinical Psychology, 26, 47-55.

Weissman, M.M., Leaf, P.J., Tischler, G.L., Blazer, D.G., Karno, M., Livingston, B.M., & Florio, L.P. (1988). Affective disorders in five United States communities. Psychological Medicine, 18, 141-153.

Weyerer, S., Hewer, W., Pfeifer-Kurda, M., & Dilling, H. (1989). Psychiatric disorders and diabetes-results from a community study. Journal of Psychosomatic Research, 33, 633-640.

Williamson, D.A., Kelley, M.L., Davis, C.J., Ruggerio, L., & Blouin, D.C. (1985). Psychopathology of eating disorders: A controlled comparison of bulimic, obese and normal subjects. Journal of Consulting & Clinical Psychology, 53, 161-166.

Wilson, G.T. (1993). Relation of dieting and voluntary weight loss to Psychological functioning and binge eating. Annals of Internal Medicine, 119, 7, 2, 727-730.

Wing, R.R. (1993). Obesity and Related Eating and Exercise Behaviours in Women. Annals of Behavioral Medicine, 15, 2-3, 124-134.

Wing, R.R., Marcus, M.D., Epstein, L.H., Blair, E.H., & Burton, L.R. (1989). Binge eating in obese patients with Type II Diabetes. International Journal of Eating Disorders, 8, 671-679.

Wing, R.R., Marcus, M.D., Blair, E.H., Epstein, L.H., & Burton, L.R. (1990). Depressive Symptomatology in Obese Adults with Type II Diabetes. Diabetes Care, 13, 2, 170-172.

Wing, R.R., Norwalk, M.P., Marcus, M.D., Koeske, R., & Finegold, D. (1986). Subclinical eating disorders and glycemic control in adolescents with type I diabetes. Diabetes Care, 9, 162-167.

Winter, W.E., et al. (1987). Maturity-onset diabetes of youth in black Americans. New England Journal of Medicine, 316, 285-291.

APPENDICES

APPENDIX A

INFORMATION AND CONSENT FORMS - DIABETIC PARTICIPANTS

INFORMATION SHEET

HEALTH AND PSYCHOSOCIAL ISSUES FOR WOMEN WITH MATURITY ONSET DIABETES

A recent survey of women with insulin-dependent diabetes in North Canterbury indicated that, for many, eating, weight control and women's health concerns are important issues. There is, however, very little information available about these issues for women with maturity onset diabetes.

This survey is to learn more about the concerns that women with maturity onset diabetes have regarding eating, weight control, mood, menstruation and contraception. We want to compare how women with and without diabetes feel about these issues. This information will help us deliver a better service to women with maturity onset diabetes, in the future.

If you choose to take part in this study, a time will be arranged for Anita or Marie to meet with you for one interview at the Diabetes Centre. You are invited to bring a friend who will be interviewed to help find out how women without diabetes feel about these issues. The interview is likely to take less than one hour. Participation in the survey will be free and transport will be arranged, if required.

After the interview, there will be some questionnaires to fill out about eating and mood, your height and weight will be measured, and a finger-prick blood sample will be taken for a glycated haemoglobin. (Your friend would not have a finger-prick done). All participants will receive a written summary of the survey findings.

If you agree to participate in the survey, your name and identity will be kept confidential, i.e. your answers will be anonymous. With your consent, your General Practitioner will be informed of your participation, and of your blood test result. If you choose not to take part, this will not affect your future treatment at the Diabetes Centre in any way. Thank you for your help and co-operation.

ANITA STROUD
Summer Research Student

PHILIPPA CAMPBELL-TIE
Clinical Psychologist/Diabetes Centre

MARIE WEBSTER
Summer Research Student

HELEN LUNT
Diabetes Physician/Diabetes Centre

SOUTHERN REGIONAL HEALTH AUTHORITY ETHICS COMMITTEE
(Canterbury) (SRHA)

HEALTH AND PSYCHOSOCIAL ISSUES FOR WOMEN WITH

MATURITY ONSET DIABETES

I have read the information sheet and have had the opportunity to discuss this study with my family and/or doctor. I understand that it has been approved by the Southern Regional Health Authority Ethics Committee.

I understand that participation is confidential (ie anonymous), and that no information will be disclosed without my permission, unless the failure to disclose information would jeopardise my safety.

I understand that I may withdraw from the study at any time, and for any reason, without adversely affecting present or future treatment.

Signature of Participant: _____ Date: _____

Signature of Investigator: _____ Date: _____

Signature of Witness: _____ Date: _____

APPENDIX B

INFORMATION AND CONSENT FORMS - CONTROL PARTICIPANTS

INFORMATION SHEET

HEALTH AND PSYCHOSOCIAL ISSUES FOR WOMEN WITH MATURITY ONSET DIABETES

A recent survey of women with insulin-dependent diabetes in North Canterbury indicated that, for many, eating, weight control and women's health concerns are important issues. There is, however, very little information available about these issues for women with maturity onset diabetes.

This survey is to learn more about the concerns that women with maturity onset diabetes have regarding eating, weight control, mood, menstruation and contraception. We want to compare how women with and without diabetes feel about these issues. This information will help us deliver a better service to women with maturity onset diabetes, in the future.

We would like to interview you to find out how women without diabetes feel about these issues so that we can compare your answers with those of women who have diabetes. If you choose to take part in this study, a time will be arranged for Anita or Marie to meet with you for one interview at the Diabetes Centre. The interview is likely to take less than one hour. Participation in the survey will be free and transport will be arranged, if required.

After the interview, there will be some questionnaires to fill out about eating and mood, and your height and weight will be measured. All participants will receive a written summary of the survey findings.

If you agree to participate in the survey, your name and identity will be kept confidential, i.e. your answers will be anonymous. If you choose not to take part, this will not affect your future treatment by Canterbury Health in any way. Thank you for your help and co-operation.

ANITA STROUD
Summer Research Student

PHILIPPA CAMPBELL-TIE
Clinical Psychologist/Diabetes Centre

MARIE WEBSTER
Summer Research Student

HELEN LUNT
Diabetes Physician/Diabetes Centre

SOUTHERN REGIONAL HEALTH AUTHORITY ETHICS COMMITTEE
(Canterbury) (SRHA)

HEALTH AND PSYCHOSOCIAL ISSUES FOR WOMEN WITH

MATURITY ONSET DIABETES

I have read the information sheet and have had the opportunity to discuss this study with my family and/or doctor. I understand that it has been approved by the Southern Regional Health Authority Ethics Committee.

I understand that participation is confidential (ie anonymous), and that no information will be disclosed without my permission, unless the failure to disclose information would jeopardise my safety.

I understand that I may withdraw from the study at any time, and for any reason, without adversely affecting present or future treatment.

Signature of Participant: _____ Date: _____

Signature of Investigator: _____ Date: _____

Signature of Witness: _____ Date: _____

APPENDIX C

BECK DEPRESSION INVENTORY (BDI)

Date: _____

_____ Marital Status: _____ Age: _____ Sex: _____

ation: _____ Education: _____

uestionnaire consists of 21 groups of statements. After reading each group of statements carefull the number (0, 1, 2 or 3) next to the one statement in each group which best describes the way yo een feeling the past week, including today. If several statements within a group seem to apply equall ircle each one. Be sure to read all the statements in each group before making your choice.

)	I do not feel sad.	
1	I feel sad.	
2	I am sad all the time and I can't snap out of it.	
3	I am so sad or unhappy that I can't stand it.	
)	I am not particularly discouraged about the future.	
1	I feel discouraged about the future.	
2	I feel I have nothing to look forward to.	
3	I feel that the future is hopeless and that things cannot improve.	
0	I do not feel like a failure.	
1	I feel I have failed more than the average person.	
2	As I look back on my life, all I can see is a lot of failures.	
3	I feel I am a complete failure as a person.	
0	I get as much satisfaction out of things as I used to.	
1	I don't enjoy things the way I used to.	
2	I don't get real satisfaction out of anything anymore.	
3	I am dissatisfied or bored with everything.	
0	I don't feel particularly guilty.	
1	I feel guilty a good part of the time.	
2	I feel quite guilty most of the time.	
3	I feel guilty all of the time.	
0	I don't feel I am being punished.	
1	I feel I may be punished.	
2	I expect to be punished.	
3	I feel I am being punished.	
0	I don't feel disappointed in myself.	
1	I am disappointed in myself.	
2	I am disgusted with myself.	
3	I hate myself.	
8	0	I don't feel I am any worse than anybody else.
	1	I am critical of myself for my weaknesses or mistakes.
	2	I blame myself all the time for my faults.
	3	I blame myself for everything bad that happens.
9	0	I don't have any thoughts of killing myself.
	1	I have thoughts of killing myself, but I would not carry them out.
	2	I would like to kill myself.
	3	I would kill myself if I had the chance.
10	0	I don't cry any more than usual.
	1	I cry more now than I used to.
	2	I cry all the time now.
	3	I used to be able to cry, but now I can't cry even though I want to.
11	0	I am no more irritated now than I ever am.
	1	I get annoyed or irritated more easily than I used to.
	2	I feel irritated all the time now.
	3	I don't get irritated at all by the things that used to irritate me.
12	0	I have not lost interest in other people.
	1	I am less interested in other people than I used to be.
	2	I have lost most of my interest in other people.
	3	I have lost all of my interest in other people.
13	0	I make decisions about as well as I ever could.
	1	I put off making decisions more than I used to.
	2	I have greater difficulty in making decisions than before.
	3	I can't make decisions at all anymore.

_____ Subtotal Page 1 CONTINUED ON BAC

THE PSYCHOLOGICAL CORPORATION
HARCOURT BRACE JOVANOVICH, INC.

I don't feel I look any worse than I used to. I am worried that I am looking old or unattractive. I feel that there are permanent changes in my appearance that make me look unattractive. I believe that I look ugly.	19	0 I haven't lost much weight, if any, lately. 1 I have lost more than 5 pounds. 2 I have lost more than 10 pounds. 3 I have lost more than 15 pounds. I am purposely trying to lose weight by eating less. Yes _____ No _____
I can work about as well as before. It takes an extra effort to get started at doing something. I have to push myself very hard to do anything. I can't do any work at all.	20	0 I am no more worried about my health than usual. 1 I am worried about physical problems such as aches and pains; or upset stomach; or constipation. 2 I am very worried about physical problems and it's hard to think of much else. 3 I am so worried about my physical problems that I cannot think about anything else.
I can sleep as well as usual. I don't sleep as well as I used to. I wake up 1-2 hours earlier than usual and find it hard to get back to sleep. I wake up several hours earlier than I used to and cannot get back to sleep.	21	0 I have not noticed any recent change in my interest in sex. 1 I am less interested in sex than I used to be. 2 I am much less interested in sex now. 3 I have lost interest in sex completely.
I don't get more tired than usual. I get tired more easily than I used to. I get tired from doing almost anything. I am too tired to do anything.		
My appetite is no worse than usual. My appetite is not as good as it used to be. My appetite is much worse now. I have no appetite at all anymore.		

_____ Subtotal Page 2

_____ Subtotal Page 1

_____ Total Score

APPENDIX D

BINGE EATING SCALE (BES)

Eating Habits Checklist

Instructions. Below are groups of numbered statements. Read all of the statements in each group and mark on this sheet the one that best describes the way you feel about any problems you have controlling your eating behaviour.

#1

1. I don't feel self-conscious about ny weight or body size when I'm with others.
2. I feel concerned about how I look to others, but it normally does not make me feel disappointed with myself.
3. I do get self-conscious about my appearance and weight which makes me feel disappointed in myself.
4. I feel very self-conscious about my weight and frequently, I feel intense shame and disgust for myself. I try to avoid social contacts because of my self-consciousness.

#2

1. I don't have any difficulty eating slowly in the proper manner.
2. Although I seem to "gobble down" foods, I don't end up feeling stuffed because of eating too much.
3. At times, I tend to eat quickly and then I feel uncomfortably full afterwards.
4. I have the habit of bolting down my food without really chewing it. When this happens I usually feel uncomfortably stuffed because I've eaten too much.

#3

1. I feel capable of controlling my eating urges when I want to.
2. I feel as though I have failed to control my eating more than the average person.
3. I feel utterly helpless when it comes to feeling in control of my eating urges.
4. Because I feel so helpless about controlling my eating I have become very desperate about trying to get in control.

#4

1. I don't have the habit of eating when I'm bored.
2. I sometimes eat when I'm bored, but often I'm able to "get busy" and get my mind off food.
3. I have a regular habit of eating when I'm bored but occasionally, I can use some other activity to get my mind off eating.
4. I have a strong habit of eating when I'm bored. Nothing seems to help me break the habit.

#5

1. I'm usually physically hungry when I eat something.
2. Occasionally, I eat something on impulse even though I really am not hungry.
3. I have the regular habit of eating foods that I might not really enjoy to satisfy a hungry feeling even though physically I don't need the food.
4. Even though I'm not physically hungry, I get a hungry feeling in my mouth that only seems to be satisfied when I eat food, like a sandwich, that fills my mouth. Sometimes, when I eat the food to satisfy my mouth hunger, I then spit the food out so that I won't gain weight.

6

1. I don't feel any guilt or self-hate after I over-eat.
2. After I over-eat, occasionally I feel guilt or self-hate.
3. Almost all the time, I experience strong guilt or self-hate after I over-eat.

7

1. I don't lose total control of my eating when dieting, even after periods when I over-eat.
2. Sometimes, when I eat a "forbidden food" on a diet, I feel like I "blew it" and eat even more.
3. Frequently, I have the habit of saying to myself, "I've blown it now, why not go all the way" when I over-eat on a diet. When that happens, I eat even more.
4. I have a regular habit of starting strict diets for myself, but I break the diets by going on an eating binge. My life seems to be either "a feast or a famine".

8

1. I rarely eat so much food that I feel uncomfortably stuffed afterwards.
2. Usually, about once a month, I eat such a quantity of food that I end up feeling very stuffed.
3. I have regular periods during the month when I eat large amounts of food, either at meal-times or at snacks.
4. I eat so much food that I regularly feel quite uncomfortable after eating, and sometimes a bit nauseous.

9

1. My level of calorie intake does not go up very high or go down very low on a regular basis.
2. Sometimes, after I over-eat, I will try to reduce my caloric intake to almost nothing, to compensate for the excess calories I've eaten.
3. I have a regular habit of over-eating during the night. It seems that my routine is not to be hungry in the morning, but to over-eat in the evening.
4. In my adult years, I have had week-long periods where I practically starve myself. This follows periods when I over-eat. It seems I live a life of either "feast or famine."

#10

1. I usually am able to stop eating when I want to. I know when "enough is enough."
2. Every so often, I experience a compulsion to eat which I can't seem to control.
3. Frequently, I experience strong urges to eat which I seem unable to control, but at other times, I can control my eating urges.
4. I feel incapable of controlling urges to eat. I have a fear of not being able to stop eating voluntarily.

#11

1. I don't have any problem stopping eating when I feel full.
2. I usually can stop eating when I feel full, but occasionally over-eat, leaving me feeling uncomfortably stuffed.
3. I have a problem stopping eating, once I start, and usually I feel uncomfortably stuffed after I eat a meal.
4. Because I have a problem not being able to stop eating when I want to, I sometimes have to induce vomiting to relieve my stuffed feeling.

#12

1. I seem to eat as much when I'm with others (family, social gatherings), as when I'm by myself.
2. Sometimes, when I'm with other people, I don't eat as much as I want to eat because I'm self-conscious about my eating.
3. Frequently, I eat only a small amount of food when others are present because I'm very embarrassed about my eating.
4. I feel so ashamed about over-eating that I pick times to over-eat when I know no-one will see me. I feel like a "closet eater."

#13

1. I eat three meals a day with only an occasional between-meal snack.
2. I eat three meals a day but I also normally snack between meals.
3. When I am snacking heavily, I get into the habit of skipping regular meals.
4. There are regular periods when I seem to be continually eating, with no planned meals.

#14

1. I don't think much about trying to control unwanted eating urges.
2. At least some of the time, I feel my thoughts are preoccupied with trying to control my eating urges.
3. I feel that frequently I spend much time thinking about how much I ate or about trying not to eat any more.
4. It seems to me that most of my waking hours are preoccupied by thoughts about eating *or* not eating. I feel like I'm constantly struggling not to eat.

#15

1. I don't think about food a great deal.
2. I have strong cravings for food but they last only for brief periods of time.
3. I have days when I can't seem to think about anything else but food.
4. Most of my days seem to be preoccupied with thoughts about food. I feel like I live to eat.

#16

1. I usually know whether or not I'm physically hungry. I take the right portion of food to satisfy me.
2. Occasionally, I feel uncertain about knowing whether or not I'm physically hungry. At these times it's hard to know how much food I should take to satisfy me.
3. Even though I might know how many calories I should eat, I don't have any idea of what a "normal" amount of food is for me.

APPENDIX E

SEMI-STRUCTURED INTERVIEW SCHEDULE - DIABETIC PARTICIPANTS

WOMEN'S ISSUES IN NON-INSULIN-DEPENDENT DIABETES

(I want to talk with you now about eating, dieting and weight control, and health issues for women, such as menstruation and contraception. I am interested in your experience and opinions of these things).

DEMOGRAPHIC INFORMATION (SUBJECTS)

Birth Date	
Age	
Ethnicity	
Height	
Weight	
HbA_1c/Random Blood Glucose	
Year of Diagnosis	
Other Medical Conditions	
Medications Other than for diabetes)	

DIABETES QUESTIONNAIRE

DIABETES MANAGEMENT

1. Have you ever been in hospital for your diabetes?

   (If Yes) What was the reason? (Diagnosis, name of hospital what year?)

2. Do you check your own blood sugar levels?

   (If Yes) How often do you check them? (Times per day, days per week?)

   How often have you checked your blood sugar levels in the last week?

   Do you use a machine meter or do you read the strips against a bottle?

EATING PATTERNS

1(a) What is the lowest weight you have been since you were 18 years old?

1(b) What is the highest weight you have been since you were 18 years old?

1(c) How satisfied are you with your current weight and body shape? VERY/MODERATELY/NOT

(If not moderately satisfied) What concerns do you have about your weight and shape?

2. Have you ever changed your eating patterns or "been on a diet" to try to lose weight? *(If Yes)* At what age did you first diet? *(Go to Question 3). (If No, go to Question 7).*

3(a) How many times in the last year (since Christmas 1993) have you changed your eating patterns or "dieted" to lose weight? *(If no diets in last year, go to Question 7).*

3(b) During the last year, what was the longest and shortest length of time you spent on a diet(s)? *(Currently on a diet?)*

4. During the diet(s), what types of changes did you make to your eating patterns? *(Specify frequency/amount/type of food or drink)*

5(a) When you were "dieting", what was your goal weight?

5(b) Did the changes result in any weight loss? *(How much weight lost and for how long?)*

5(c) Overall, is your weight any different to what it was a year ago? *(If Yes, how?)*

6. Did you notice your blood sugars going low on a diet? If so, how did you know your blood sugars were low? *(That is, symptoms/SMOBG).* What did you do about it?

7. Are you worried that your blood sugars may go too low if you diet?

(If Yes) Does this affect your approach to dieting?

(If not currently dieting) Are there any particular worries or reasons which stop you from dieting?

8. Have you ever seen a dietitian? *(When/how often?).*

(If No), go to Question 9. (If Yes), When was the last time you saw a dietitian?

How helpful was this last appointment? *(NOT/MODERATELY/VERY)*

In what way was the last appointment unhelpful/moderately helpful/very helpful?

What changes to your eating patterns did the dietitian recommend?

Which of these are you doing now?

What has stopped you from making the other changes that were recommended?

Would it be helpful to see a dietitian again?
YES/NO/PERHAPS/DON'T KNOW. If unhelpful, why?

9. Do you think that having diabetes would affect a person's weight? YES/NO

(If Yes) In what way?

10(a) Are you on tablets or insulin to help control your blood sugars?
If no oral hypoglycaemics/insulin, go to Question 16.
(If Yes) What is the name of the tablets?/What sort of insulin are you taking?

10(b) What is your recommended dose for these tablets/the insulin?
Number/Units? Frequency?

11. What dose of the tablets/insulin are you actually taking at present?

(Note any variation from recommended dose)

(Sometimes miss out on taking tablets/insulin?)

(If different to recommended dose) Why do you not always take the recommended dose?

12. Do you think that your diabetes tablets/insulin affect your weight?

(If Yes) How do they affect your weight? INCREASE/DECREASE

13. Have you ever thought about altering the dose of your diabetes tablets/insulin to change your shape or weight? *If No, go to Question 16.*

(If Yes) What have you thought of doing? INCREASE/DECREASE

14(a) Have you ever actually altered the dose of your diabetes tablets/insulin to try to change your shape or weight? (INCREASE/DECREASE/AMOUNT)

(If Yes, to 14(b); If No, to 14 (c).

14(b) Did you tell your doctor you altered the dose of your tablets/insulin to change your shape or weight?

(To Question 15)

14(c) What made you decide not to alter your tablets/insulin?

(To Question 16)

15. Have you altered the dose of your diabetes tablets/insulin to change your shape or weight in the last month?

(If Yes) How many times have you changed your tablets/insulin in the last month?

By what amount?

16. Are there any other ways you have tried to control weight, get rid of food you had eaten, change your body's shape? (Exercise, pills, vomiting?)

APPENDIX F

SEMI-STRUCTURED INTERVIEW SCHEDULE - CONTROL PARTICIPANTS

WOMEN'S ISSUES IN NON-INSULIN-DEPENDENT DIABETES

(I want to talk with you now about eating, dieting and weight control, and health issues for women such as menstruation and contraception. I am interested in your experience and opinions of these things).

DEMOGRAPHIC INFORMATION (CONTROL GROUP)

Birth Date	
Age	
Ethnicity	
Height	
Weight	
Other Medical Conditions	
Medications	

DIABETES QUESTIONNAIRE

EATING PATTERNS

1(a) What is the lowest weight you have been since you were 18 years old?

1(b) What is the highest weight you have been since you were 18 years old?

1(c) How satisfied are you with your current weight and body shape? VERY/MODERATELY/NOT

(If not moderately satisfied) What concerns do you have about your weight and shape?

2. Have you ever changed your eating patterns or "been on a diet" to try to lose weight? *(If Yes)* At what age did you first diet? *(Go to Question 3). (If No, go to Question 7).*

3(a) How many times in the last year (since Christmas 1993) have you changed your eating patterns or "dieted" to lose weight? *(If no diets in last year, go to Question 7).*

3(b) During the last year, what was the longest and shortest length of time you spent on a diet(s)? *(Currently on a diet?)*

4. During the diet(s), what types of changes did you make to your eating patterns? *(Specify frequency/amount/type of food or drink)*

5(a) When you were "dieting", what was your goal weight?

5(b) Did the changes result in any weight loss? *(How much weight lost and for how long?)*

5c) Overall, is your weight any different to what it was a year ago? (If yes, how?)

7. *(If not currently dieting)* Are there any particular worries or reasons which stop you from dieting?

8. Have you ever seen a dietitian? *(When/how often?).*

(If No), go to Question 9. (If Yes), When was the last time you saw a dietitian?

How helpful was this last appointment? *(NOT/MODERATELY/VERY)*

In what way was the last appointment unhelpful/moderately helpful/very helpful?

What changes to your eating patterns did the dietitian recommend?

Which of these are you doing now?

What has stopped you from making the other changes that were recommended?

Would it be helpful to see a dietitian again?
YES/NO/PERHAPS/DON'T KNOW. If unhelpful, why?

9. Do you think that having diabetes would affect a person's weight? YES/NO
(If Yes) In what way?

16. Are there any other ways you have tried to control weight, get rid of food you had eaten, change your body's shape? *(Exercise, pills, vomiting?)*

APPENDIX G

CODING SYSTEM - DIABETIC PARTICIPANTS

Diabetic participants

Coding system

- Years since diagnosis **(number)**
- Main method of diabetes control **1 insulin**
  2 tablets
  3 insulin + tablets
  4 diet only
- Medical Problems **1 yes**
  0 no

thyroid, depression, other psychiatric illnesses, heart (eg: angina), cholesterol, high blood pressure, hypertension, asthma, other (see appendices)

1 yes
0 no

- Medications **1 yes**
  0 no
- Number of times hospitalised for diabetes **(number)**
- self SMOBG **1 yes**
  0 no
- how many times in last week **(number)**
- machine meter **(1)** or strips against a bottle **(0)** or both **(2)**
- lowest weight (since 18 years old) **(kg)**
- highest weight (since 18 years old) **(kg)**
- range of weight change (since 18 years old) **(kg)**
- satisfied with current weight and body shape **1 yes (very)**
  2 (moderately)
  3 no (not)
- concerns with current weight and body shape **1 yes**
  0 no
- decrease weight **1**
- increase weight **0**
- other concerns **0 health; 1 clothing; 2 bone structure; 3 others comments; 4 more toned/shape concerns; 5 more stable weight desired; 6 low self-esteem + confidence**
- ever dieted **1 yes**

0 no

- age of first diet **(years)**
- number of diets in the last year **(number)**
- shortest time spent on a diet in the last year **(weeks)**
- longest time spent on a diet in the last year **(weeks)**
- main change made to diet (in the last year)

  1 eat less
  2 eat less fat
  3 less protein
  4 more balanced diet
  5 eats one meal per day
  6 decrease carbohydrate intake
  7 increase salads
  8 decrease sweet consumption
  9 increase carbohydrates
  10 regular meals

- goal weight when dieting **(kg)** **0 Don't know**
- any change in weight - increase **1**, decrease **0**, don't know **2**, or same **3**
- how much **(kg)**
- other changes
- weight change maintained **1** yes **0** no
- approximately how long for **(weeks)**
- weight different to last year **1 yes**
  0 no
  1 increased
  0 decreased
- Were blood sugars low on a diet? **1 yes**
  0 no
  2 DK
- How did you know? **1 symptoms**
  0 SMOBG
- action taken

1 increase sugar, 2 nothing (good level), 3 monitor more closely

- Worry that blood sugars may go too low on diet? **1 yes**
  0 no
- Does this effect dieting approach? **1 yes**

0 no

- action taken

  1 increase carbohydrates and snacks between meals

  2 monitor food intake

  3 eat sensibly and monitor glucose levels

  4 don't diet

Non-dieters (in last year)

- Reasons for not dieting **1 yes**
  0 no

Specific reasons

- not needed **1 yes**
  0 no
- other reasons

1 not happy when dieting; 2 miss foods; 3 body change, illness; 4 if unwell, 5 periods irregular and decreases energy; 6 pointless/frustrating; 7 lack of willpower; 8 low blood sugars; 9 damages health; 10 financial constraint, 11 diabetes, 12 self-defeating, 13 worry if I don't stay on it, 14 family commitments, 15 lifestyle (eg: too busy), 16 boring, 17 mood decreases - eat more, increased pressure, 18 added pressure.

- Seen a dietitian before **1 yes**
  0 no
- helpful? **1 yes**
  0 no
- does diabetes affect a persons weight **1 yes**
  0 no
  2 DK
  increases it **1**
  decreases it **0**
- other

2 big/overweight people; 4 obesity/overweight = risk factor; 5 get 'fat or thin'; 6 lifestyle and eating changes; 7 causes weight fluctuations; 8 they are small people, 9 forced to eat if low sugars, 10 harder to loose the weight, 11 has maintained my weight, 12 determined by

attitude to food and lifestyle, 13 metabolism slowed up, 14 increased appetite, 15 need to lose it for diabetes management.

- Recommended dose of insulin/tablets
- Always take recommended dose **1 yes**
  0 no
- How change -increase **(1)** or decrease **(0)**
- other - **1 increase/decrease according to blood sugars and exercise, 2 i/d according to what eaten, 3 forgets/confused/busy/can't be bothered, 4 if away, 5 if had alcohol, 6 can't afford to, 7 doctors advise**
- medication affect weight **1 yes**
  0 no
  2 DK
- increase **(1)** or decrease **(0)** or **(2)** both - increase insulin and decrease tablets.
- Have you ever thought about altering medication dose to change weight etc.

  1 yes 0 no

  1 increase 0 decrease 2 both
- ever done it? **1 yes**
  0 no
- action **1 increase, 0 decrease, 2 both**
- tell doctor? **1 yes**
  0 no
- why not alter **1 complications unattractive**
- Have you altered your medication <u>in the last month</u> to change your shape/weight?

  1 yes 0 no
- How many times? **(number)**
- By what amount? **(number)**
- other weight control methods ever tried
- pills **1 yes**
  0 no
- vomiting **1 yes**
  0 no
- exercise **1 yes**
  0 no

APPENDIX H

CODING SYSTEM - CONTROL PARTICIPANTS

Control participants

Coding System

- Medical Problems **1 yes**
  0 no

thyroid, depression, other psychiatric illnesses, heart (eg: angina), cholesterol, high blood pressure, hypertension, asthma, other

1 yes
0 no

- Medications **1 yes**
  0 no
- lowest weight (since 18 years old) **(kg)**
- highest weight (since 18 years old) **(kg)**
- range of weight change (since 18 years old) **(kg)**
- satisfied with current weight and body shape **1 yes (very)**
  2 moderately
  3 no (not)
- concerns with current weight and body shape **1 yes**
  0 no
- decrease weight **1**
- increase weight **0**
- other concerns **0 health; 1 clothing; 2 bone structure; 3 others comments; 4 more toned/shape concerns**
- ever dieted **1 yes**
  0 no
- age of first diet **(years)**
- number of diets <u>in the last year</u> **(number)**
- shortest time spent on a diet in the last year **(weeks)**
- longest time spent on a diet in the last year **(weeks)**
- main change made to diet (in the last year)
  1 eat less
  2 eat less fat
  3 less protein
  4 more balanced diet
  5 eats one meal per day
  6 decrease carbohydrate intake
- goal weight when dieting **(kg)** **0 Don't Know**

- any change in weight - increase **1**
  - decrease **0**
  - Don't know **2**
- maintained weight loss **1 yes**
  0 no
- for how long **(weeks)**
- weight different to last year **1 yes**
  0 no
- increased **1**
  decreased **0**

Non-dieters (in last year)

- Reasons for not dieting **1 yes**
  0 no

Specific reasons

- not needed **1 yes**
  0 no
- other reasons

1 not happy when dieting; 2 miss foods; 3 body change, illness; 4 if unwell, 5 periods irregular and decreases energy; 6 pointless/frustrating; 7 lack of willpower; 8 low blood sugars; 9 damages health; 10 financial constraints.

- Seen a dietitian before **1 yes**
  0 no
- helpful? **1 yes**
  0 no
- does diabetes affect a persons weight **1 yes**
  0 no
  2 DK
- increases it **1**
- decreases it **0**
- other possibilities

2 big/overweight people; 4 obesity/overweight = risk factor; 5 get 'fat or thin'; 6 lifestyle and eating changes; 7 causes weight fluctuations; 8 they are small people.

- other weight control methods ever tried
- pills **1 yes**
  0 no
- vomiting **1 yes**
  0 no
- exercise **1 yes**
  0 no

APPENDIX I

OTHER MEDICAL PROBLEMS - DIABETIC PARTICIPANTS

Diabetic participants
Other medical problems

2 Directiculitis
11 Arthritis
1 Scleroderma
4 Back problems
1 Raynards disease
1 Muscular dystrophy
1 Muscular atrophy
1 Blindness
2 Deafness
1 Emphysema
1 Irritable bowel syndrome
1 Bowel hernia
1 Hiatus hernia
1 Priniceous anaemia
1 Bronchitis
1 Fluid retention
2 Stroke
1 Ulcerated leg
4 Ulcers'
1 Cystoneurea
2 Epilepsy
1 Excema
1 Rhinitis
1 Hayfever
1 Multiple Sclerosis
1 Psoriasis

APPENDIX J

OTHER MEDICAL PROBLEMS - CONTROL PARTICIPANTS

Control participants
Other medical problems

1 Hearing problem
1 Sinus problem
1 'Nutcracker gullet'
2 Minniers disease
1 Back problem
3 Arthritis or Rheumatoid Arthritis
2 Kidney reflux
1 ME
1 Hayfever
1 Degenerative cervical spine
1 Migraine headaches
1 Hiatus hernia
1 Factor-7 bleeding disorder
1 Stomach ulcer
1 Perforated eardrum

APPENDIX K

OTHER PSYCHIATRIC ILLNESSES - DIABETIC PARTICIPANTS

Diabetic participants
Other psychiatric illnesses

1 anxiety
1 bipolar disorder
1 panic attack's